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CONTENTS

Endocrine and Fatigue Headaches. *Neil Campbell Stevens* 801

Observations on Sweating. *Frederick M. Allen.* 806

Preliminary Note on the Mode of Action of Irradiated Ergosterol. *Walter Bauer* and *Alexander Marble* 809

Entero-Intoxication. *Lloyd Arnold* 811

Synthalin-B in Diabetes Mellitus. *Helmuth Ulrich* 814

Trichiniasis with Widespread Infestation of Many Tissues. *S. Seymour Horlick* and *Ralph E. Bicknell* 816

MEDICAL PROGRESS

Progress in Tuberculosis, 1928-1929. *John B. Hawes, 2nd,* and *Moses J. Stone* 820

VERMONT STATE MEDICAL SOCIETY

House of Delegates, 1929 828

Communicable Disease Problem. *Charles F. Dalton* 829

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Case 15431—Albuminuria and Unexplained Sepsis. *Chester M. Jones.* Case 15432—A Suitable Case for Treatment with Quinidine Sulphate. *Howard B. Sprague* 833

EDITORIALS

Socialization of Medicine 842

Resumption of the Campaign for Raising Money for the Boston Medical Library and the Massachusetts Medical Society 842

More Care in Criticism of Hospitals in Order 843

This Week's Issue 843

(Contents continued on next page)

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PATHOLOGY DEPARTMENT

CONTENTS

(Continued)

BOOK REVIEWS

The Diagnosis of Pancreatic Disease. Robert Coope. 856
Experiments and Observations on the Gastric Juice and the Physiology of Digestion. William Beaumont 855
The History of Nursing. James J. Walsh..... 856
An Introduction to Pharmacology and Therapeutics. J. A. Gunn..... 856
Minor Surgery. Frederick Christopher..... 855
Pathogenic Micro-organism. William H. Park; A. W. Williams; and Charles Krumwiede..... 856
The Principles of Clinical Pathology in Practice. Geoffrey Bourne and Kenneth Stone..... 856

CORRESPONDENCE

Information Regarding Opportunities for General Practice. A. W. Stearns..... 848
Dr. Chevalier Jackson of Philadelphia to Lecture at Brockton. Thomas H. McCarthy..... 848
Parking Privileges in New York. Frank R. Ober... 848
A Plan Whereby Patients Can Be Sure of Service. Samuel B. Woodward..... 847

MISCELLANY

Comparison of Disease Incidence in Connecticut with 1928 and Seven Year Average Month Ending September 28, 1929..... 847
Dr. Cushing Speaker at Johns Hopkins..... 844
Lead Poisoning..... 819
Massachusetts Hospitals Approved by the Hospital Standardization Conference of the American College of Surgeons..... 846
Medical Center Project Outlined..... 832
Methods of Detecting the Presence of Carbon..... 841
The Middlesex Health Association..... 846
New Officers of Delta Omega..... 832
Rhode Island Mortality Statistics: 1928..... 844
Testimonial Dinner to Dr. Leary..... 846
Two Fellowships for the Wilmer Ophthalmological Institute 844
Undulant Fever Is Increasing..... 846

NEWS ITEMS..... 849
NOTICES 850
OBITUARIES 849
RECENT DEATHS..... 848
REPORTS AND NOTICES OF MEETINGS..... 850
SOCIETY MEETINGS, CONGRESSES AND CONFERENCES 854

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ENDOCRINE AND FATIGUE HEADACHES*

BY NEIL C. STEVENS, M.D.†

THIS paper deals with types of headache which have heretofore been classified as functional, namely: migraine, nervous or fatigue headache, muscular tension headache, menstrual headache, etc. To apply the term functional to this group is neither accurate nor scientific. The definition of "functional disease" is a "disorder affecting the functions of an organ as distinguished from the substance of an organ." Under the old classification, the headache of Bright's disease is put in the organic group and migraine is classed as functional; yet the headache of Bright's disease is as truly functional as migraine. The headache of nephritis is probably due to the accumulations of toxins in the nervous system; the headache of migraine is likewise due to the accumulation of poisonous products in the nervous system. In neither case is there any demonstrable lesion in the brain; in both cases the headache is dependent on a disturbance elsewhere in the body. Is it not an arbitrary distinction to call one organic and the other functional? Again, the headache occurring at the time of the menstrual period has been called functional. In many of these cases, the uterus is of the infantile type, the menstrual flow nearly always scanty. The headache is dependent upon an organic cause just as much as the headache of nephritis is dependent upon a lesion of the kidneys.

Since the etiology of these headaches is pretty definite, why not give them an etiological classification? As this paper will endeavor to show, some of the headaches in this group result from an endocrine deficiency and others from fatigue, both mental and physical. So we have classified them under "Endocrine and Fatigue Headaches" and have divided them into five groups as follows:

(1) Migraine
(2) Menstrual migraine
(3) Nervous or fatigue headache
(4) Muscular tension headache
(5) Headache of the menopause.

MIGRAINE

Migraine is one of the most characteristic symptom complexes in medicine. It is a hereditary, constitutional disease, characterized by periodic unilateral headache, disturbance of vision, nausea and vomiting. Migraine usually begins between the ages of 8 and 16. The hereditary element can be traced in nearly all cases It is seldom seen among the laboring classes.

Etiology

Most of the old theories as to the causation of migraine have been abandoned. There are only three at the present time that are worthy of consideration:

The first is that migraine is due to an intestinal toxemia or an auto-intoxication. Undoubtedly there are many attacks, the severity and duration of which are diminished by a thorough evacuation of the bowel at the onset of the attack. But this explanation is unsatisfactory as it does not take into consideration the strong hereditary element, or explain satisfactorily the periodicity of the attack. Neither does it explain why the toxemia should appear in the first or second decades and disappear at the menopause.

That migraine is anaphylactic in nature is a conception that has been seriously advanced. Sufferers are supposed to be sensitive to some food, which, when ingested, will precipitate an attack. It is hard to explain by this theory, as it is by the toxaemic theory, the marked periodicity of the attacks in many cases, why, if the attack is dependent upon the intake of a certain food, it should occur regularly at a certain hour of a certain day of the week. In a study of over 50 cases of migraine at the Cornell Clinic, food has played an important part in only three. In one of these an orange would precipitate an attack; in another, beefsteak was the exciting cause and in a third, shell-fish was the etiological factor. But in none of these would the elimination of the particular food prevent attacks of migraine. They would make their appearance sooner or later, regardless of diet. At the beginning of this study, sensitization skin tests were made routinely, but the results did not lend support to the theory of anaphylaxis and they were later discontinued.

Although we began the study of migraine, four years ago, at the Cornell Clinic, with no preconceived notions as to its cause, we are forced to the conclusion by a considerable variety of evidence that the causality lies deep-seated in the germ plasm, that it is an hereditary, constitutional disease, and that it seems to be

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†For record and address of author see "This Week's Issue," page 842.

associated closely with a dysfunction of the endocrine glands.

The evidence in favor of an endocrine etiology is as follows—first, the age of onset. Migraine usually begins at the onset of menstruation, in girls, or just before and at the beginning of adolescence in boys. It ceases in women at the menopause, or changes its character. It is rare to see it in men over 50 years of age. Menstrual migraine is identical with true migraine symptomically, except that it occurs only once a month, and has undoubtedly some relation to the menstrual period. It is invariably associated with a scanty menstrual flow. In a series of sixty cases of migraine in men and women, the basal metabolic rate was estimated. In the male group, half the cases studied (numbering ten) were below normal, ranging from —11 to —24. The rest of the group was within the limits of normal. In women, almost without exception, the basal metabolic rate was either low normal or subnormal, ranging from —2 to —16. The majority of these cases give other evidence of a hypothyroid condition, such as subnormal temperature, sensitiveness to cold, fatigue, etc. The favorable response to treatment with glandular extracts also tends to support this theory.

Assuming that hypofunction of the thyroid is responsible for many cases of migraine, it might be permissible to suggest an explanation of the cause of an attack, by outlining what occurs in the body during and following an attack of migrainous headache. The thyroid gland furnishes a hormone which regulates the metabolism of the body and which is intimately connected with the nervous, circulatory and digestive systems. If there is an insufficient amount of hormone, the bodily processes are slowed, the mentality is sluggish, the pulse becomes slow, the desire for food is diminished. Is it not possible that the food consumed is incompletely burned and that the poisonous products of incomplete combustion accumulate in the system and, when the limit of tolerance is reached, precipitate an attack? An explosion occurs with its expression in the symptom complex of migraine; the thyroid undergoes a temporary stimulation under which, for a short time, it puts out more than its ordinary amount of hormone; the bodily processes are stimulated (a temperature of 102 or 103 often occurring during an attack), the toxaemia is eliminated, and the organism quickly returns to its normal state. (Patients often assert that they never feel so well as they do immediately following an attack.) The thyroid relapses to its former condition of low output and the cycle begins again, to end, in a week or two, in another explosion. It may be asked why this is not the old theory of toxaemia re-stated. It is, but it is also an explanation of the toxaemia and of the periodicity of the attacks and the quick recovery.

Fortunate indeed are the sufferers from migraine who permanently escape the zeal of the nose and throat specialists. The story of the removal of tonsils, septums, turbinates, the opening of sinuses and antrums is all too common. The extraction of wisdom teeth is the special ambition of dentists. The opthalmologists tell you that astigmatism and eye strain cause a large percentage of migraine cases. There is very little excuse for these mistakes. Migraine is a definite entity. It is one of the most characteristic of symptom complexes. It begins early in life, usually at puberty, and ends rather abruptly between the fourth and fifth decades. The attack itself comes at intervals. It is usually violent, with intense, one-sided headache, accompanied by nausea and vomiting and usually visual disturbances. It could hardly be mistaken for anything else even with the most superficial history-taking and yet fully 30% of patients who enter the Cornell Clinic with this complaint have been operated on by nose and throat men. I know of no cure resulting from surgery.

Heredity

Some authors state that heredity is a factor in fully 90% of cases of migraine. In one series of cases, 70% give a history of migraine or sick headaches in either father or mother. In families with a neurotic strain, it is more apt to occur. The close association between migraine, epilepsy, asthma, allergy and eczema has been often noted. In some cases it is difficult to decide whether one is dealing with petit mal or the incipient stages of migraine. At its beginning, migraine is often characterized by transient attacks of vertigo without headache and only later declares itself by the typical hemicrania.

Symptoms of migraine: Migraine may begin as early as five years of age. The usual time of onset is at adolescence, or just before. Headaches that simulate migraine, but which begin some years after puberty, are not uncommon. These cases, with late onset and a symptom complex in some instances almost identical with that of migraine, I believe should not be classified in the true migraine group. In their history the hereditary element is lacking. They lack the typical periodicity; the attacks are usually milder and they respond with comparative ease to treatment. In the above classification, we have put them into the nervous or fatigue group of headaches. Foci of infection in tonsils, teeth, gall bladder or appendix, toxic conditions, constipation, nervous strain and tension, anything which tends to depress the vitality of the body, is liable to result in this type of headache. It is this group, which so closely resembles migraine and is so easy to cure, that accounts for such theories of etiology as disease of the gall bladder and stasis in the duodenum, two of the latest theories. No doubt proper elimination and the removal of a diseased gall bladder have cured

many cases of headache, but they were not migraine.

So, in the diagnosis of migraine, the invariability of the early onset at adolescence or before must be clearly appreciated. That is a rule which allows of few exceptions.

Although in some few cases the onset is heralded by vertigo and nausea, which, as we have said before, resemble very closely the beginning of epilepsy, in about 98% the onset is characterized by headache. Pain is the prominent symptom: first a feeling of fullness, then a dull headache localizing in the orbit, in the temple, or over the temporal bone. The pain gradually increases in severity until it reaches its maximum in a few hours. In the great majority of cases, the pain is accompanied by nausea and by vomiting, which is often violent and incessant. In many cases the sufferer is prostrated and is totally unable to perform any work. Some patients look forward to spending two days out of every fortnight in bed. Great importance has been attached by the older writers of the visual disturbances of migraine. They made elaborate charts of the visual fields and the scotomata were described with the utmost detail. The phenomena are undoubtedly interesting but of not much importance. They seem to be merely an indication of an extreme toxic condition.

Premonitory symptoms of an attack differ. Most patients complain of drowsiness and lethargy, some of chilliness and extreme fatigue. They usually know pretty well when an attack is coming on.

Attacks of migraine may occur once a week, once in two weeks or once a month. In its beginning, attacks occur less frequently. A young girl or boy may experience only two or three during the first year, a half dozen during the second. These gradually increase in frequency and intensity until a cycle peculiar to the individual is reached. This cycle is maintained, with slight irregularity, for the next twenty-five to thirty years, when it gradually diminishes and automatically ceases between the ages of 48 and 55. It is rare to see a case of migraine in men over fifty years of age. It is apt to persist longer in women.

Prognosis

In the majority of cases, migraine ceases between the ages of 48 and 55, with or without treatment. If the attacks do not stop then, they usually change their character; the headache becomes more frequent and less severe.

The occupation, the environment, the state of health of the individual have much to do with the frequency and severity of the attacks. An outdoor life is of utmost benefit to an individual suffering from migraine.

Treatment of Migraine in Women

The treatment of migraine in women varies somewhat from that of men, so the two will be discussed separately. Women do not respond as well to treatment as men. There are other factors with which to deal. There is a large nervous element, which complicates most of the cases of migraine in women. A woman who is subject to migraine may have attacks of headache which closely simulate migraine but which are of nervous origin and which are cured by the removal of the inciting cause. The total number of cases studied intensively were 29. They have been under observation from three months to four years. Out of 29, 14 were improved (it is dangerous to make the claim that any of them have been cured), 5 were unimproved, 10 could not be followed over a long enough period to justify any conclusion as to the value of the treatment. The basal metabolic rate was estimated in all. In 27 cases it varied from —2 to —27. Two were on the plus side of normal, plus 2 and plus 17.

Every effort was made to improve the general health. Fresh air and sunlight are valuable adjuncts to treatment; it seems to me that sunlight has a distinctly beneficial effect. Injections of iron, arsenic and strychnine are of value in many cases. Thyroid, in doses of one to three grains a day, seems to help a considerable number. Mixed gland, when the basal metabolism is normal, is often more effective. Luminal is used freely in the beginning, in doses of grains ½, three times a day, to eliminate the nervous element.

Migraine in Men

Migraine shows very much the same characteristics in males that it does in females. It has its onset either before adolescence or puberty. It is rarely seen beyond the age of 50. There is the same hereditary tendency, the same periodicity of the attacks, the hemicrania associated with nausea and vomiting. It may or may not be preceded by an aura or by other unpleasant sensations. The malady is even more clear-cut and definite than it is in the opposite sex. In a great many cases of migraine in women, there are other factors, neuroses for example, which make the condition complicated and the treatment difficult. The nervous element is a minor factor in males, in whom we have to deal with only migraine itself.

A total of 26 cases has been studied. In 20 the basal metabolic rate was estimated. These were equally divided into two groups, one with a subnormal basal metabolism and one with a normal rate. In the subnormal group, the metabolism ranged from —11 to —24. The average was —18. The remaining ten fell within the limits of normal, ranging from —8 to plus 12. In 6 of the 26 cases, the basal metabolic rate was not estimated. The majority of the first group gave definite evidence of hypothyroidism: sluggishness in three, defect of memory, sensitiveness to cold, dry skin, falling out of hair,

subnormal temperature, etc. These symptoms were not common to the second group. In fact, the difference symptomatically between these two groups is striking.

Of all types of headache we have treated, this group has given the most satisfactory results and the response to treatment has been rapid. In more than half the headache has disappeared entirely. Of 26 cases that have been followed over a period of three months to three years, 18 have been improved or very much improved, 3 unimproved. Five have failed to return to report their condition. And these results were obtained among a class of patients, who, for the most part, had been seeking relief for years.

The hypothyroid group is treated with thyroid, the dose varying from one to three grains a day. Members of the second group, those with a normal basal metabolism, do not respond to thyroid. At the beginning of this investigation, both groups were treated alike with thyroid. In some of them there was an immediate favorable response and in others, the symptoms were aggravated. In those that reacted unfavorably, it was noted that the basal metabolism was not subnormal. Mixed gland was tried empirically and the results were equally as good as in the hypothyroid group.

For over a year different glandular substances were experimented with. Pituitary was tried according to the theory of Timme. In only one case has it proved very successful. For some months the adrenal extract alone was used, with no success at all. In my opinion, with little scientific evidence to substantiate it, the testicular element in mixed gland is responsible for its beneficial action.

MENSTRUAL MIGRAINE

The most common form of migraine is that which occurs at the time of the menstrual period. It comes on either before or after the period, sometimes both before and after. It is extremely punctual in the time of its appearance, the patient being able to foretell the day of onset exactly.

The attack is usually severe and is nearly always associated with nausea and vomiting. Most women are unable to pursue their usual occupations and are forced to remain in bed a day or two. The headache lasts from six to twenty-four hours.

Menstrual migraine is nearly always associated with a scanty menstrual flow. We have yet to notice this condition in a girl or woman where the flow was profuse. Many women are under the impression that their periods are normal when they flow three days and soil two or three napkins on the day they flow the most. For the examining physician merely to inquire whether the catamenia is normal is not enough. Menstrual migraine seems to bear some relation to dysmenorrhoea, often appearing to be a substitute for it, although both conditions may occur in the same individual. Both, in all probability, depend upon an endocrine dysfunction and the consequent lack of development of the uterus.

The writer of this article for a long time was extremely dubious of any treatment for this condition. Nothing but failure was recorded for several years. The reason for this failure was that the treatment was not sufficiently prolonged. Ovarian extract, including the whole gland, if given over a period of a year or two, will produce a marked amelioration in the symptoms. Most patients take the gland for a month or two and, not deriving an apparent benefit from it, discontinue it. But if one can get the co-öperation of the patient and induce her to give it a year's trial, surprising results will be obtained.

If, as it is generally conceded, the development of the uterus is dependent upon the hormone of the ovary, it is not to be wondered at that no results are evident in one to three months' time after beginning treatment. It will take a much longer period than that to stimulate the growth of the uterus.

In the course of two or three months after treatment has been instituted, there is usually cessation of nausea and vomiting; the pain is not diminished to any considerable degree. Then there will come a month when there is no attack of pain or vomiting, which fills the patient's mind with a false hope of cure. The following month she may have a severer attack than ever. But, gradually, periods unaccompanied by attacks become more frequent, until the old conditions are reversed and the majority of the menstrual periods are passed by with very little discomfort. I cannot say that any case yet in my series has been completely cured. Occasionally there will come a month when the old trouble will return. These upsets are usually due to fatigue, strain or nervousness.

In the treatment of this condition, exercise is of the utmost importance, particularly those exercises directed to the development of the muscles of the abdomen and thighs. This condition, we have noticed, is extremely rare among women who lead an outdoor life. Among women of the peasant class it is almost unknown. Pregnancy and child-birth often diminish the severity of these attacks.

My practice has been to give whole ovary grains V, three times a day, and continue its administration as long as the symptoms last.

NERVOUS HEADACHE

Nervous headache is seen more commonly in women than in men. It may make its appearance at any age, most commonly in the third decade. It may be caused by worry, excitement, anger, fatigue and nervous strain. There are

probably very few adults living who, at one time or another, have not suffered from this type of headache. In the high-strung type of individual, it is a very common complaint. It is a frequent accompaniment of migraine, especially in women. With the exception of headache due to constipation, it is the most common of all types. It may occur every day or two, or three times a week. Unlike migraine, it is irregular in its appearance, following fatigue or excitement. It follows so closely upon any excess that the sufferer is usually aware of the cause. This headache is liable to become a habit with the individual and if the conditions which produce it remain unchanged, the headache is apt to increase in frequency and severity. Any focus of infection which lowers vitality predisposes the individual to nervous headache. Married women in the third or fourth decade, who are underweight and overworked, are most subject to nervous headache.

The site of the pain is variable. The usual site is upon the vertex, especially among older women. They complain more of pressure than of pain. In others the occiput is commonly attacked. It may, like migraine, be in one eye or temple. With each attack the same place is involved.

It is a condition easy to diagnose and it usually responds well to treatment. Of therapeutic measures, rest and freedom from worry and nervous strain are the most important. It is often cured by luminal alone. Nervous headache occurring in asthenic individuals, who are easily fatigued, is often cured by thyroid alone.

MUSCULAR TENSION HEADACHE

Muscular tension headache seems to be a more common complaint of women than of men. In all probability it occurs among the latter, but we have yet to see at the clinic a case in a male. It is not uncommon in women. It is a characteristic entity and its differentiation from other types of headache is simple. The pain begins in the back of the neck or along the upper border of the trapezius. It then involves the occiput and in a severe attack radiates to the forehead along both sides of the head. The neck becomes stiff and rotation of the head is painful. On palpation of the upper border of the trapezius, exquisitely tender spots can be elicited and the muscle feels tense and contracted. The patients complain of a drawing sensation at the back of the neck, as if the head was being pulled back. Fatigue, worry, high nervous tension are the commonest causes. It is common among stenographers and among those whose work forces them to hold their arms and shoulders in certain positions for long periods. It is analogous to a writer's cramp.

Treatment

Most of these cases improve with treatment. Rest and relaxation are important. Some people hold their muscles tense while engaged in their daily activities. After a while, it becomes a habit and they are tense even while resting. When this practice is long continued, a fatigue state supervenes and the individual complains of fatigue all the time. To effect any permanent cure of this condition, patients must learn the secret of physical and mental relaxation. Of physical measures, gentle massage of the muscles of the shoulders and neck is often helpful. Heat is beneficial. Luminal in small doses aids in producing relaxation.

HEADACHE OF THE MENOPAUSE

This headache is common in women between the ages of forty-five and fifty-five. It is more usual in those who formerly had migraine, although none of the peculiar characteristics of migraine remain. The headache is only occasionly severe. It is dull in character, occurring daily and lasting most of the day. The patient usually complains of it on arising. It is aggravated by worry and fatigue. The site of the pain may be frontal, occipital or upon the vertex. It is most commonly in the latter location. There is often more of a sense of pressure than of pain. In this group the headache is often not the prominent symptom, but one of many causing discomfort, a sense of inadequacy, nervousness and fatigue.

There are, at this period, two distinct groups in which headache occurs, the hypothyroid group and that group associated with the classical disturbances of the menopause. It is important to recognize this distinction as the treatment is diametrically opposite. The hypothyroid group is characterized by a dull headache, fatigue, lack of energy, subnormal temperature, low blood pressure, slow pulse, dry skin, a sensitiveness to cold and a low basal metabolism. The second group complains of hot flashes, nervousness and headache. In this group, there has been no instance where the basal metabolism has been subnormal. For this type ovarian extract acts almost as a specific, while thyroid or sometimes mixed gland acts with almost equal success in the first. In the first group there is a tendency to anemia and the general condition is often benefited by the hypodermic injection of iron and arsenic. Luminal is often useful.

Reference — New England Journal of Medicine, July 25, 1929, page 168.—"The Thyroid and Headache at the Menopause."

OBSERVATIONS ON SWEATING*

BY FREDERICK M. ALLEN, M.D.†

SWEATING has long been employed for therapeutic purposes, but elimination by this method has not attained any high degree of demonstrated value. Though it is known that sweat contains some of the principal constituents of the urine, a significant substitution of the skin for the kidney function in nephritis has not been established. A slight benefit from sweating in acute nephritis may be admitted as probable, though no reduction of the blood nitrogen has been proved. In chronic nephritis the value of sweating stands as negligible; no reduction of nitrogen retention has been shown, and there is no serious claim that patients are kept alive for any long time by sweating after extreme failure of the kidney function.

Inasmuch as the prognosis, when uremia is not controllable by diet regulation, is now hopeless, and as vicarious function of the skin seems to be the only possible means of aid, the question was presented whether this function can be employed more effectively. Patients with renal-vascular disease often react poorly to sweating; the quantity of perspiration may be small, or the resulting prostration may outweigh the supposed benefit. In the presence of this impaired sweating capacity, the methods which have been employed heretofore seem to be calculated to produce the least good and the most harm. Perspiration has been induced by drugs, which are necessarily brief and depressing in their application, or by heating methods which surround the body with an atmosphere saturated with vapor, or which in other ways tend to confine heat in the body.

With due consideration of the standards of "effective temperature" established by heating engineers, it remains an apparent fact that heat can be endured most comfortably and with least prostration when there is low humidity and sufficient ventilation. In the dry air sweating serves its natural purpose, namely, the cooling of the body. Up to high limits, increased heat is balanced by increased perspiration and evaporation. These familiar and neglected principles seem worth a trial for prolonging the duration and lessening the debility of sweating.

Mere climatotherapy has accomplished little in nephritis, because patients are free to seek the coolest spots and the time and degree of sweating are not subject to regulation. Our equipment for imitating or improving upon a desert atmosphere, developed during the past two years, consists of a so-called heat chamber, divided into two parts for the two sexes, each part being a room more than 20 feet square with an 8 foot ceiling and an adjoining bathroom. Adequate ventilation with dry air enables most persons to live in such a chamber at temperatures from 140 to 180 degrees Fahrenheit, without serious discomfort. Treatment for 24 hours daily has been tried, but the best results at present seem to be obtained with a duration not longer than 12 to 14 hours per day, allowing the patient to sleep in a cool room. In suitable cases there is no difficulty in thus producing 4 litres of sweat per day for an indefinite number of days without prostration. Also in suitable cases the blood chemistry and clinical condition are modified in a manner never before demonstrated.

In addition to the nephritic cases, we are using the heat chamber for rheumatic infections, other forms of arthritis, neuritis, certain skin troubles and superficial infections, peripheral arteriosclerotic conditions, etc. We are also proceeding to the use of dry air, warmed or cooled to suitable degrees, for a variety of respiratory infections and other disorders. Comparative analyses of the sweat in normal and diseased states are being accumulated with the aid of R. I. Wagner. Accurate collection of unaltered sweat is recognized to be difficult. Our device consists of a broad sheet of rubber dam, with sufficient absorbent cotton inside it, secured tightly to the back by bands passing around the chest. The patient reclines in a chair covered by a towel. His weight makes close contact of rubber and skin to prevent evaporation, and the towel absorbs sweat from the surrounding areas so it cannot run inside the rubber. The usual collection is from an area of 80 square inches for 1 hour.

Although this method is at least sufficiently accurate to demonstrate marked variations in the composition of the sweat, we have as yet no basis for a general law governing the function of the sweat glands. One of the important known factors is the blood chemistry; for example, the concentration of urea and creatinin is apt to be high in the sweat when it is high in the blood. Any close parallelism is upset by numerous discrepancies and variations, which may theoretically be due to the large or small quantities of perspiration in a given time, the amount of water in the body, the availability of the water (for example in edemas easy or hard to clear up), the supply of sodium chloride or other diaphoretic substances, the efficiency of the circulatory apparatus, states of the nervous system, the number and specific activity of the sweat glands themselves and skin conditions modifying these, and constitutional states such as fever, thyroid disorders, etc. One result of the observations is to confirm the view that a large proportion of renal-vascular cases are characterized by deficiency in sweating or in the

*From The Physiatric Institute, Morristown, New Jersey.
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†For record and address of author see "This Week's Issue," page 843.

vascular reaction to heat. This abnormality is commonest in cases with severe hypertension, but may also be found in other cases, especially in the terminal stages. Some of these patients begin to sweat but develop severe collapse, so that sweating treatment is not feasible by any nephritic cases is widespread throughout the body and not limited merely to the kidneys. The cases which respond best to sweating are presumably those in which the renal factor is more prominent than the general vascular disorder. We have therefore wished to make trials

TABLE I

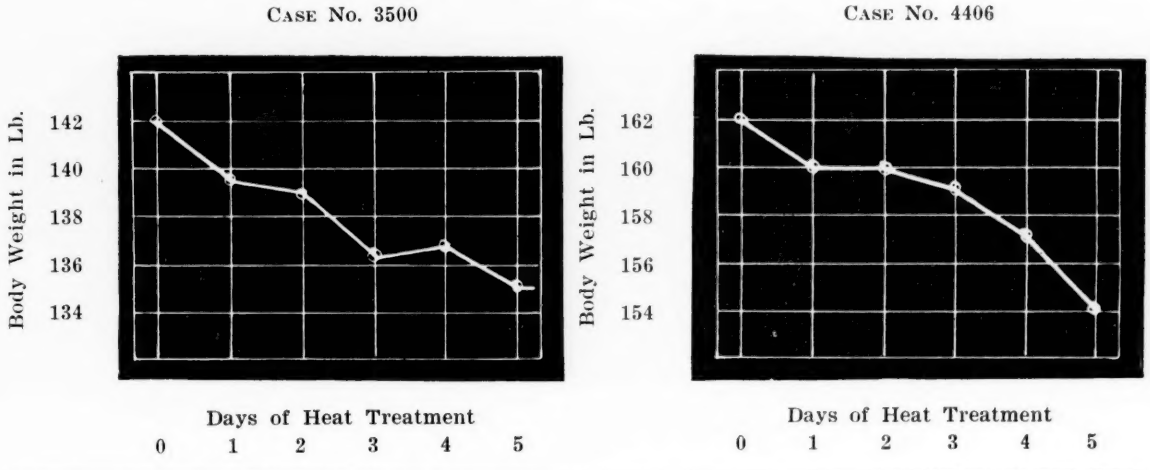
COMPARISONS OF UREA, CHLORIDE AND CREATININ IN BLOOD AND SWEAT

Date	*Blood* Urea mg. %	*Blood* Plasma Chloride mg. %	*Blood* Creatinin mg. %	*Sweat* Urea mg. %	*Sweat* Sodium Chloride mg. %	*Sweat* Creatinin mg. %	Wgt. Lb.	Method of Collection
1928				CASE 24. *Uremic Coma, Diabetes.*				
12-28	134	523	12.0	1740	128	11.7	128	*Collected from skin
1929								
1-1	132	499	15.2	1420	102	9.7	133	Ditto
1929				CASE 3500. *Nephritis, Hypertension.*				
3-1	58	606	5.0	185	160	2.1	133	Rubber and cotton
3-8	56	606	4.0	90	95	2.0	135	" " "
3-24	66	560	4.0	86	159	<1	135	" " "
4-9	76	515	4.3	113	159	<1	135	" " "
4-19	88	495	4.5	205	205	1.6	137	" " "
4-22	82	507	3.9	143	95	0.9	133	" " "
5-4	82	457	5.7	195	124	1.1	135	" " "
				CASE 4406. *Nephritis, Myocarditis, Hypertension.*				
3-23	56	495	3.3	94	60	<1	159	" " "
3-26	40	523	4.0	90	41	<1	156	" " "
				CASE 4974. *Essential Hypertension—Cardiac Decompensation.*				
3-6	34	540	—	50	188	<1	151	" " "
3-23	24	549	1.4	60	94	<1	146	" " "

*The sweat running in rapid streams from cleansed areas of skin was allowed to flow into a test-tube. Some evaporation is possible owing to the absence of covering.

CHART I

REDUCTION OF EDEMAS (5 DAYS HEAT TREATMENT)



means whatever. Other patients subjected to heat respond not with sweating but with fever, so that the attempt must be abandoned. The difficulty is, apparently, not a mere cardiac weakness, for some severe heart cases may respond well to sweating. The observations support the view that the vascular disorder in many with acute nephritis, anuria from any cause, mercury poisoning, etc., in which we may encounter maximal kidney impairment with practically normal circulation and sweating power, but unfortunately we have not had any of this class. The chronic cases in which sweating cannot be endured are necessarily hopeless. Some

other patients can tolerate only very brief sweating by any method. But our observations show that some chronic cases are of a type which respond satisfactorily to intensive sweating treatment.

Table I presents some simultaneous analyses of blood and sweat, illustrating the partial parallelism in a few of the principal constituents, but also the lack of any complete correspondence. It is also evident that the concentrations of urea and chloride, in particular, must represent an important quantitative excretion when reckoned on 3 or 4 litres of sweat daily. Incidentally, this chloride excretion is sufficient to prevent carrying out any prolonged intensive sweating treatment with salt-free diet. The automatic protection of the salt reserve by the kidneys does not hold good for the sweat glands, and under these circumstances the salt ration must be precisely regulated in order to prevent deficiency symptoms, on the one hand, or damage from excessive salt on the other. It may be noticed that the sweating obtained by previous methods has never accomplished enough elimination to require these precautions.

TABLE II

INFLUENCE OF SWEATING UPON THE BLOOD VOLUME (REDUCTION OF HYDREMIA)

	Before Treatment	After 6 Days Heat Treatment	
Body Weight	143 lb.	135 lb.	— 6%
Total Blood Vol.	6060 cc.	5340 cc.	—13%
Plasma Vol.	4570 cc.	3875 cc.	—18%
RBC Volume	1480 cc.	1465 cc.	0%

CHART II

INFLUENCE OF SWEATING UPON BLOOD UREA AND PLASMA CHLORIDE IN UREMIA

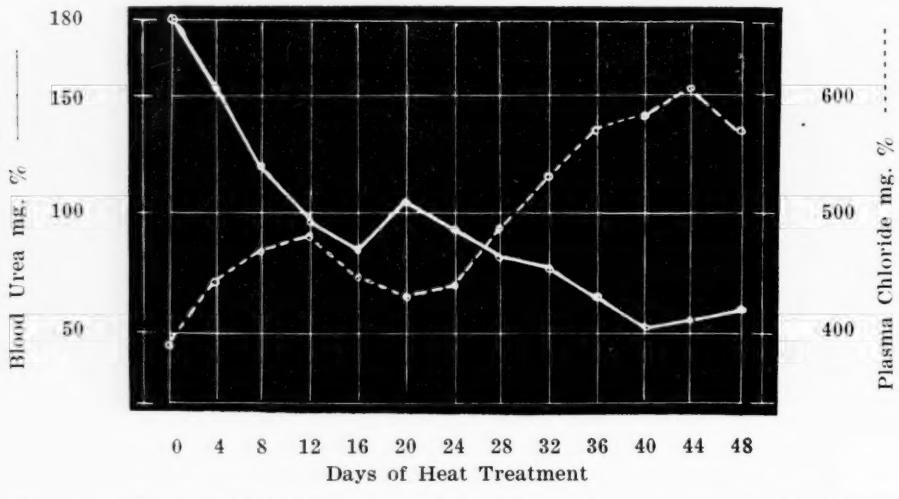


TABLE III.

Case No. 3500. Age 34 Years. Male

Blood					Urine					Diet	
Date 1929	Urea mg. %	Creatinin mg. %	Plasma Chlor. mg. %	Indican	Vol. cc.	Albumin (Esbach)	T.N. gm.	Indican	Blood pressure	Protein gm.	NaCl Wgt. lb.
1-28*	180	5.4	391	+++	1235	1.8		++	130/86	20	0 132
2-1	158	7.2	441	++	345	1.1		+	130/80	"	0 128
2-7	98	7.5	487	+++	195	0/6	1.09	+++	128/78	"	0 129
2-17	92	5.0	483	++	1150	2.1	3.22	++	124/86	"	4 132
2-24	72	4.0	597	+	600	1.3	2.24	++++	174/100	"	4 132
3-2	58	5.0	606	+	650	0.9	2.22	++++	150/92	"	4 133
3-9	56	4.0	606	0	580	2.5	1.92	+	178/100	"	4 134

* Heat treatment begun.

Edema may also be influenced by the sweating treatment. In certain cases it seems to be less efficient than diuretics, but in other cases it succeeds in reducing or removing edema when drugs, including Salyrgan, are unsuccessful or contraindicated. The sweating treatment is especially valuable in cases of edema combined with high blood nitrogen, when other methods often entail difficulty or danger.

Table II shows an instance of reduction of hydremia by sweating.

In this same connection, attention may be called to the behavior of the plasma chlorides in certain conditions (Table III, Chart II). It is interesting that the low plasma chloride, which

is a significant abnormality in many uremic cases, rose while the urea fell, though both urea and chloride were being eliminated through the skin.

Table III presents more complete analyses in a case of advanced uremia under the sweating treatment. This patient was a business man, aged 31 years when first seen in November 1925; his chronic nephritis was evidently of some years' standing at that time. Up to 1928 the disease became progressively more severe because of habitual departures from diet, and during 1928 the strict adherence to a salt-free diet with only 20 gm. protein daily could no longer control the rising blood nitrogen. The patient was brought to the Institute on January 27, 1929, after a typical uremic attack of muscular twitching and unconsciousness. In our former experience, cases reaching this stage, after a chronic progression uncontrolled by the strictest diet, have regularly ended fatally within a brief time. This patient readily tolerated 10 or 12 hours of sweating daily, during which he drank and eliminated through perspiration about 4 litres of water each day. The analyses indicated that on some days more urea was eliminated through the sweat than through the urine. Also, on sweating days the urine was diminished in both water and solids, as compared with days without sweating, and this diminution could not be prevented by the utmost encouragement of drinking. This reduction of work was interpreted as rest for the kidneys, and there has seemed to be a chance that the kidney function might recover to some extent through such rest. The clinical condition improved along with the blood chemistry. The patient became able to leave the Institute and resume business. At home, under diet treatment alone, his condition has tended to become gradually worse. On salt-free diet the blood urea has risen. The giving of 1 to 4 gm. of salt daily to stimulate diuresis of nitrogen has resulted in edema and hypertension. At present this man comes to the Institute for week-ends of sweating, and carries on his regular business the remainder of the time.

CONCLUSIONS

In conclusion, it must be recognized that the cases studied up to the present have been few, and it is still impossible to say what proportion of cases may be controllable by this means. The observations seem to furnish the first definite evidence of benefit and prolongation of life and comfort by sweating in certain forms of renal-vascular disease. If any results are obtainable by sweating, it is believed that the method described represents an improvement over those formerly used.

PRELIMINARY NOTE ON THE MODE OF ACTION OF IRRADIATED ERGOSTEROL*

BY WALTER BAUER, M.D.,† AND ALEXANDER MARBLE, M.D.†

THE use of irradiated ergosterol in the treatment of certain disorders of calcium metabolism has resulted, in suitable cases, in very prompt and striking improvement[1,2,3,4,5,6,7]. A full appreciation of the results obtained with this relatively new vitamin D concentrate forces one to realize its therapeutic advantages, as well as the harmful effects which may be produced by injudicious use of this potent drug. The literature pertaining to irradiated ergosterol is already voluminous, although it has only been a few years since its discovery. Nevertheless, a review of this literature leaves one with very little data which adequately explain its mode of action. That hypercalcemia can be produced by the use of irradiated ergosterol has been reported by various workers[1,2,4]. However, a serum calcium value tells only the height of the "calcium stream" existing in the blood at a given time, and gives no indication of its direction of flow. Only by a study of the total calcium metabolism can one obtain the necessary information which shows the direction of flow of the "calcium stream"—either from the bones into the excretory channels, or from the gastro-intestinal tract towards the bones. The beneficial use of irradiated ergosterol in diseases caused by failure to absorb calcium, such as infantile tetany, rickets and osteomalacia[1,2,3,4,5,6,7] hints that it is of distinct aid in the assimilation of calcium for bone deposition.

Several theories have been suggested to explain the mechanism of action of irradiated ergosterol:

1. That it is stored in the body[1,17].
2. That it causes increased calcification of bone by drawing calcium from the non-osteoid tissues[8,9].
3. That it acts as a stimulus to the parathyroid glands[1].

The first of these theories has some proof[17], but even if the administration of the drug caused an increased body store of irradiated ergosterol, we are still left without an explanation of its mode of action.

The second hypothesis scarcely seems reasonable because the body tissues, other than bones, contain very little calcium. (Muscle and liver contain approximately 10 milligrams of calcium per 100 grams of tissue.) The experiments of

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†For record and address of author see "This Week's Issue," page 843.

Hess et al.[9] at first glance seem to offer sufficient proof that the calcium necessary to produce the hypercalcemia (after irradiated ergosterol administration) is derived from the tissues. Closer examination of their article, however, suggests two valid objections to their interpretation of data obtained. In the first place, their experimental period in which metabolism studies were made (three days) does not allow adequate time for the mineral metabolism of an animal to become stabilized. In the second place, their statement that the intestinal tract becomes free of calcium after three days on a low calcium diet is improbable. Unpublished data from this laboratory show that the calcium normally excreted via the bowel is re-absorbed when irradiated ergosterol is being administered.

The third theory, that irradiated ergosterol (in therapeutic doses) acts as a stimulus for the parathyroid glands, appears to be an unlikely interpretation, for all the data thus far presented would indicate that the hypercalcemia resulting from parathyroid extract injections is at the expense of the bones[10,11]. The effects following the administration of an active parathyroid extract are always similar, whether the dose is small or large (unless toxic doses are given). Careful metabolism studies have shown that the injection of a potent parathyroid extract results in:

1. A fall in the serum phosphorus.
2. An increase in the urinary phosphorus excretion. (The fecal phosphorus excretion is little, if at all, affected.)
3. A rise in the serum calcium.
4. An increased calcium excretion. This increase is urinary, since the calcium excretion in the feces is unaffected.

That the increased calcium and phosphorus excretion is at the expense of the bones has been proved by animal experimentation, as well as by study of diseases of the parathyroid gland in man. The prolonged administration of parathyroid extract to rabbits results in marked decalcification of the bones, easily demonstrable by x-ray examination[12], or by macroscopic examination of cross-sections of the same bones. Case reports of Mandl[13], Barr, Bulger and Dixon[14], and Bauer, Aub and Albright[15] demonstrate that hyperactivity of the parathyroid glands in man results in marked decalcification of the bones. Furthermore, hypoparathyroidism presents changes which are the reverse of those following parathormone administration, as outlined above. With the knowledge that the parathyroid hormone pulls calcium and phosphorus from the bones, any explanation suggesting that the action of irradiated ergosterol in therapeutic doses is secondary to parathyroid stimulation would seem unreasonable, because its therapeutic employment results in *increased* calcification.

Since the hypercalcemia and increased calcification which follow the administration of irradiated ergosterol cannot be explained by any of the above theories, one is forced to seek some other interpretation as to its mode of action. Other than the bones, the only source of calcium and phosphorus is the gastro-intestinal tract. It is reasonable to suppose that irradiated ergosterol may act, as does cod liver oil and ultraviolet light[16], by increasing the absorption of these elements by this route.

Assuming that its mode of action might be such, the following experiments were undertaken. Knowing from previous work on cats[12] that a high calcium diet replaces bone trabeculae lost during a preceding period on a low calcium intake, we wished to ascertain whether the addition of irradiated ergosterol to a high calcium diet would hasten the restoration of these trabeculae. Accordingly, twelve cats were placed on a low calcium diet for a length of time sufficient to show a marked diminution of the bone trabeculae. At the end of this period, the left forelegs of all the cats were amputated at the shoulder under ether anesthesia. Following this, all twelve cats were placed on a high calcium diet; six of the twelve were given irradiated ergosterol daily.* The animals were sacrificed in pairs (one control animal and one receiving irradiated ergosterol), at varying lengths of time following institution of the high calcium diet regime, and the humeri of the right forelegs were saved for examination. Therefore, we have for comparison the left and right humerus of each animal. The left humerus represents the effect of the low calcium diet in all twelve cats; the right humerus represents in six of the animals the effect of the high calcium diet alone, and in the other six the effect of the high calcium diet plus irradiated ergosterol. The bones were all prepared as previously described[12]. Although their calcium intake was far less (approximately from 1/5 to 1/2 that of the controls), the cats receiving irradiated ergosterol showed a much greater increase in the number of trabeculae in the humeri than did their normal control mates.

These findings would seem to suggest the conclusion that irradiated ergosterol increases the amount of calcium which is absorbed from the gastro-intestinal tract. If this increased absorption occurs at a time when the body needs calcium, there results an increased deposition of calcium and an increase in the number of trabeculae. Complete metabolism studies (unpublished data) on patients in this clinic prove conclusively that there is a marked increase in calcium absorption when irradiated ergosterol is administered.

These findings make it apparent why the use of irradiated ergosterol in the treatment of infantile tetany, rickets, and osteomalacia has met with such uniformly beneficial results. They are all disorders of calcium metabolism in

*We are greatly indebted to the Winthrop Chemical Company for the large amounts of irradiated ergosterol ("Vigantol") with which they supplied us.

which the mechanism at fault is absorption from the gastro-intestinal tract.

Hamilton[16] suggested that a normal child has a calcium store at birth which can be drawn upon during the first four months of life. Similarly it has been shown in kittens that their bones are filled with trabeculae at birth, only to disappear as growth takes place[12]. The beneficial results obtained from the use of irradiated ergosterol as a prophylactic agent against rickets might easily be explained in this manner. Its administration at the time of need results in a greater absorption of calcium and an increase in the readily available supply of calcium, thus reducing the likelihood of the child's developing rickets.

In a later report we shall present in detail these and other studies concerning the effect of irradiated ergosterol upon the blood and total calcium metabolism and any pathological changes observed. Certain evidence at present suggests that the primary action of the drug may be in altering the composition of the pancreatic juice. Studies are now in progress in an attempt to prove or disprove this point.

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ENTERO-INTOXICATION*

BY LLOYD ARNOLD, M.D.†

THE mucous membrane of the gastro-intestinal tract is a covering layer of the body. We are accustomed to regard this epithelial lining as a specialized digesting and absorbing structure. There is a definite obligate bacterial flora in the lumen of the gastro-intestinal tract, the components of this bacterial community differing at various levels of the tract. The bacterial flora of the large intestine has received considerable attention and the predominance of a proteolytic type of bacteria has been associated with auto-intoxication due to the absorption of certain split-products of proteins. This type of flora can be changed to a fermentative type by diet. We wish to call attention to the upper end of the small intestine as a port of entry of bacteria and bacterial products that can cause an intoxication of the organism.

The bacteria swallowed by mouth are not added to the flora of the large intestine. There must, therefore, be a bacterial destroying mechanism present in the gastro-intestinal tract. It was thought that the acid in the stomach was the main factor involved in the destruction of the ingested bacteria. The acidity of the stomach, however, is an untrustworthy barrier against the entrance of bacteria into the intestinal tract. Bartle and Harkins[1], Cornwall and Le Frenais[2] and Kestner[3] have recently reviewed this question and have substantiated earlier observations. Schuetz[4] showed in dogs that certain bacteria placed in the duodenum were destroyed before reaching the ileum. Rolly and Liebermeister[5], using the rabbit, substantiated these observations. Eiskamp and Park[6] demonstrated that bacteria injected into tied-off segments of the small intestine in dogs were destroyed in a short period of time. Ricen, Sears and Downing[7] found the duodenum of all healthy subjects to be entirely free from any organisms except those having well-known aciduric properties. Arnold and Brody[8] associated the bactericidal power of the upper part of the small intestine with the acid-base balance of the contents of the lumen of this part of the alimentary tract.

In the animals so far investigated it has been found that the upper half of the small intestine is usually slightly acid in reaction. McClendon, Bissel, Lowe and Meyer[9] have shown this to be true for several animals, including man. Under normal conditions the duodenum of the horse, the cow[10], rat, guinea-pig and rabbit[11], and

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in fact most animals, seems to have a predominance of hydrogen over hydroxyl-ion. The only source of acid is the gastric secretion; the secretions below the pylorus are alkaline or alkaline buffered. Under physiological conditions there must be a co-ördinated mechanism controlling the amount and probably the acid-base contents of the secretions entering the upper levels of the gastro-intestinal tract in order to maintain the slightly acid reaction of the duodenum and jejunum.

Moro[12] found a fecal type of bacterial flora in the duodenum of infants suffering from summer diarrhea. This author thought the infant was intoxicated by the absorption of bacterial products from the upper part of the small intestine. The normal infant has few bacteria in this part of the alimentary tract; usually only enterococci were found. Moro's term "endogenous intoxication" was based upon these observations. Moro and his co-workers[13] also called attention to the difference in the reaction of the duodenum and upper jejunum in the infants suffering from diarrhea. The reaction changed from slightly acid to alkaline. These investigators pointed out that under normal conditions the alimentary tract was acid down to the lower jejunum or ileum, where a neutral or alkaline reaction was first encountered. Few bacteria were present in the acid reacting level, but a rich fecal type of bacterial flora was present in the neutral or alkaline segments of the tract.

Van der Ries[14] has recently reviewed the literature upon the hydrogen-ion concentration and bacterial flora of the small intestine. Most investigators have observed an increase in the bacterial flora of the duodenum and upper jejunum when there was a diminution in the hydrochloric acid secretion of the stomach. In an experimental study of this problem in dogs, Arnold and Brody[15] found that by injecting an alkaline buffered solution into the duodenum, the bacterial flora could be changed to the type usually encountered in the ileum and caecum. We observed that at the same time there was a change in the concentration of leucocytes in the peripheral circulation. The leucocytes tended to accumulate in the peripheral capillary beds as a result of duodenal alkalinization. In view of the recent advances made in the knowledge of autonomic status of the various functional systems of the body, we are inclined to think that our experiments came within this field of work. Petersen[16] has briefly reviewed this subject.

A peripheral leucocytosis is associated with a parasympathetic status of the skin and adjacent tissues. Under these conditions there is an increase in the functional activity of the skin, as an organ. Accompanying this increase in the physiological activity of the peripheral area of the body, there is a sympathetic status in the splanchnic system. The gastro-intestinal tract, liver and other splanchnic glands have a diminished number of leucocytes per cubic millimeter of blood and the physiological function is inhibited, or opposite to that of the hyperactive peripheral system. Petersen and his coworkers have considered the sympathetic and parasympathetic equilibrium between the internal organs and the superficial body areas as a "peripherosplanchnic balance". Investigations have been made in regard to changes in this peripherosplanchnic equilibrium in relation to vascular permeability (Mueller and Petersen[17]); gastric motility and secretion (Mueller and Petersen); biliary secretion (Mueller and Kast[18]); gastric tonus (Arquin[19]).

When an animal is responding to external stimuli in a normal manner, that is, when the physiological units of the body are interacting in a co-ördinated functional way, such an animal can be considered normal or in a state of good health. If an animal cannot adapt its body activities to the external stimuli, then dysfunction becomes manifest and such an animal is not a healthy or normal organism. The external stimuli are received upon some body surface; skin, upper respiratory tract, gastro-intestinal tract, specialized sensory organs, etc. Climatic changes, such as temperature, humidity, wind velocity, sun-light, barometric pressure, etc., are of prime importance as external stimuli. Food and all ingested substances act as stimuli upon the gastro-intestinal tract. If the animal can adapt itself to its meteorological and nutritional environment, then the parasitic factors are of less importance than when the organism is in a state of maladjustment to its environment. This brings us to the consideration of the periphero-splanchnic balance, as mentioned before. This term does not introduce anything new, but this conception helps us to crystallize our ideas upon some of the changes that take place within the body during the process of adaptation to our ever-changing environment. When an animal is placed in a warm and humid environment, there is an increase in the skin activity and a decrease in the physiological functions of the splanchnic region. The stomach contracts and secretes less acid, the biliary secretion is diminished, the hepatic function as a whole is diminished (thoracic duct lymph analysis), the splanchnic region becomes sympathetic in its orientation (Peterson, et al). Arnold and Brody[8] have shown that the upper end of the small intestine loses its power to destroy ingested bacteria under these conditions. These same workers[20] have shown that bacteria can be demonstrated to be present in the thoracic duct lymph stream after intraduodenal injection, if there is a sudden disturbance in the periphero-splanchnic balance. There is no evidence of permeability of the wall of the intestinal tract for bacteria unless there is a sudden change in the equilibrium between the splanchnic and peripheral systems of the animal.

There is a change in the distribution of the endogenous bacterial flora in the small intestine in the experimental animal after the ingestion of an alkalinized food material, during the pe-

riod of adaptation to a high external temperature environment, during fever as a result of an infection and also as a result of fever produced by foreign protein injection (Arnold[21]). It has been shown, in connection with the heat-stable toxins of the Salmonella group of food-poisoning bacteria, that by changing the temperature of the environment, one can obtain varying degrees of inhibition of the bactericidal power of the intestinal tract (Arnold and Singer[22]). These observations were extended to include experimental diarrhea in dogs (Arnold and Hull[23]). The first detectable change in the distribution of the intra-intestinal bacterial flora is noted in the lower segments of the jejunum. The contents of this part of the gut, instead of showing a transition between upper jejunum and ileum, has a typical ileal flora and the middle part of the jejunum represents the transitional zone. The duodenum and upper jejunum are the last to lose the power to regulate the bacterial flora within their lumen. Under some experimental conditions the ileal flora extends up to the pylorus and even into the stomach. We can reproduce in the dog, under proper experimental environments, the "endogenous infections" Moro described for infants.

Members of the B. coli family are seldom present in the normal endogenous flora of the upper half of the small intestine (Van der Ries). They form a part of the flora of the large intestine. When there is an ascension of the flora from the lower levels into the upper part of the alimentary tract, the B. coli group is found in the duodenum and upper jejunum. Stuart, working in this laboratory, has demonstrated two toxic substances in sterile filtrates of B. coli cultures. When broth cultures of B. coli are incubated twenty-four to forty-eight hours and passed through a Berkefeld filter, the filtrate causes convulsions and death when injected subcutaneously into rabbits. When broth cultures of B. coli are incubated for fourteen days and passed through Berkefeld filters, the filtrate, injected subcutaneously into rabbits, causes diarrhea and death. The young cultures seem to have toxic substances that act upon the nervous system, the old cultures, toxic substances that act upon the intestinal tract. The former we have called "exotoxins" and the latter "endotoxins" for purposes of convenience in nomenclature. Sublethal doses of these toxic substances do not produce increased resistance to a subsequent lethal dose of the same toxic material. These observations substantiate the earlier findings of Vincent[24]. Stuart found that small doses of the older culture filtrates, injected at weekly intervals into rabbits, would cause a chronic diarrhea with gradual loss of weight and death of the animal after several weeks. The same filtrate given per os, every other day for three weeks, would lead to diarrhea and loss of weight. If the oral administration was continued, death followed. The autopsy showed a chronic colitis in the rabbits that had received the sterile filtrates of the fourteen-day old B. coli cultures; the subcutaneous, intravenous and per os administration had the same end results. He was unable to produce any demonstrable changes in rabbits by the oral administration of the filtrates of young B. coli cultures (Arnold and Stuart[25]).

We attempted experimentally to place B. coli products of metabolism in the lumen of the intestinal tract in small doses over a period of several weeks. This led to the same abnormal symptoms and pathological findings as when the intravenous or subcutaneous route was used for the administration of this toxic material. If there was an interference with the normal distribution of the endogenous bacterial flora of the alimentary tract with an ascension of the B. coli group into the duodenum and upper jejunum, it is possible that by-products of these bacteria would be absorbed under these conditions in a manner similar to that seen in our experiments after oral administration. The upper part of the small intestine is an absorbing area; ordinarily it is relatively free of bacteria. We have found that when the bacteria from the large intestine ascend into the upper intestinal levels, there is a change from a slightly acid to a neutral, or even an alkaline, reaction. It should be borne in mind that calcium and some other chemical substances are soluble only in an acid medium; hence a change in reaction has an important influence upon body metabolism. Telfer[26] has emphasized the importance of the acid-base equilibrium of the absorbing part of the small intestine in relation to calcium and phosphorus metabolism.

Auto-intoxication from the large intestine is an explanation for certain symptom complexes generally accepted by medical men. It is possible that much of the intoxicating material is absorbed through the upper part of the intestinal tract. When the gastric acid secretion is diminished, as in the older age group where constipation, colitis, "auto-intoxication", etc., are most prevalent, the food is not properly acidified in the stomach. This gradually leads to a change in the reaction of the upper part of the small intestine. The bile, pancreatic juice and succus entericus are alkaline fluids. Unless the stomach saturates the food with acid and shows an excess of free acid above the saturation point, there is danger of a change in the acid-base equilibrium of the lumen of the specialized digesting and absorbing levels of the duodenum and jejunum. When this takes place, there is an ascension of the fecal flora into these highly differentiated physiologically active upper intestinal levels.

The purpose of our experimental work has been to try to understand some of the factors underlying host susceptibility to gastro-intestinal diseases, particularly as this was influenced by climate and diet. This subject has been recently reviewed in the light of our experience (Arnold[27]). The question of the influence of

our work upon the present conceptions of intoxications of intestinal origin was thought worthy of recording.

The decrease in the functional response of the gastric mucosa to secrete acid with advancing age is well brought out by the studies conducted by Keefer and Bloomfield[28], Friedenwald and Morrison[29], and others. If the stomach does not properly acidify its contents, the margin of safety in maintaining a proper acid-base equilibrium within the lumen of the upper half of the intestinal tract is reduced to a minimum. This would be reduced still farther if food were ingested requiring a large amount of acid to neutralize its alkaline content. The ingestion of a food requiring a minimum amount of gastric acid secretion should be advantageous. The pediatrician has been forwarding this idea for some time. The "Eiweiss-milch", protein-milk, lactic acid milk and other forms of acidified milk are based upon the principle of insuring a proper acid-base equilibrium within the lumen of the upper half of the small intestine (Arnold[30, 31]). It does not seem unreasonable to extend the same biological principle to persons in the age group over forty years who show a failure of gastric response to food. This latter age group possesses greater powers of adaptation and does not show the acute intestinal disturbances so characteristic of the infant. The process is a gradual diminution in gastric acidification. The replacement of this acid deficiency by diet should aid the organism in maintaining a normal intra-intestinal acid-base content. Soured milk in its various forms should be indicated. Citrus fruits are acid in reaction and contain an alkaline residue after absorption. It is seldom realized that lemon juice requires about an equal amount of one normal alkali for its complete neutralization. It should not be forgotten that acidophilus milk, bulgaricus milk and other therapeutic procedures to produce the so-called "transformation" of the intestinal, or rather the fecal, flora are in fact acid-buffered dietetic principles.

SUMMARY

Experimental evidence has been cited to show that the upper part of the small intestine can play an important part in systemic intoxication usually associated with the term "auto-intoxication". Changes in the distribution of the endogenous bacterial flora of the alimentary tract have been discussed. Certain alterations in the diet have been suggested to aid the body in the process of adaptation to the changes in gastro-intestinal function.

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SYNTHALIN-B IN DIABETES MELLITUS*

BY HELMUTH ULRICH, M.D †

TAKING cognizance of the fact that guanidin poisoning causes hypoglycemia, Frank, Nothmann and Wagner[1] began a series of studies to determine whether or not this property of guanidin could be utilized in the treatment of diabetes. They found that the introduction of additional methyl groups in the formula of guanidin had a tendency to reduce its toxicity without diminishing its power to lower blood sugar. The compound they evolved was decamethylin-diguanidin, which later received the commercially patented name, synthalin.

Many reports concerning the use of synthalin in the treatment of diabetes mellitus may be found in the German medical literature. The drug has one great advantage over insulin: it is active when taken by mouth. Its disadvantages are its relative feebleness of action and a tendency to elicit such symptoms of poisoning as icterus, nausea, vomiting, diarrhea and headache. The drug is believed to have a deleterious effect on the liver, but the damage appears to be transitory.

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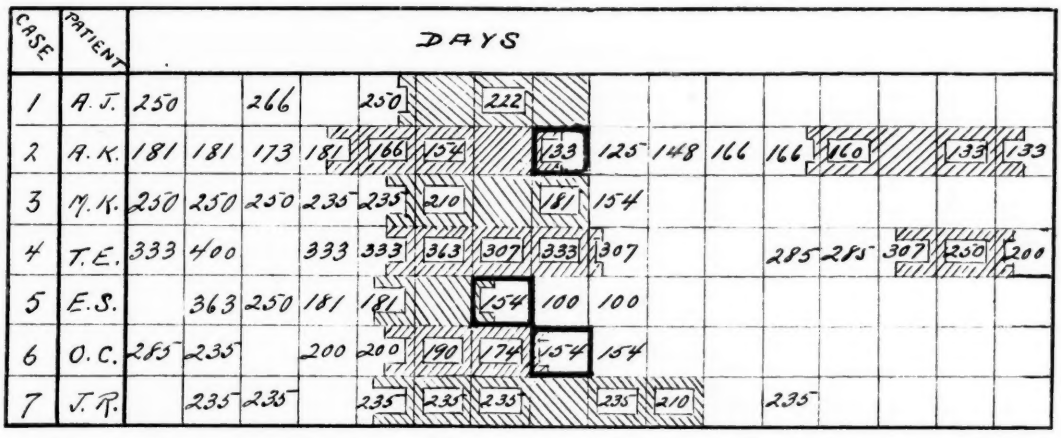
More recently, Frank and his collaborators, in an effort to reduce toxicity further, have modified the formula of synthalin by introducing a still greater number of methyl groups. They gave to the improved product the name *neosynthalin* and later *synthalin-B*. Reports concerning it are few.

The present study deals with the effect of treatment with synthalin-B on the level of blood sugar of patients with pancreatic diabetes. The amount used in each case was 40 milligrams a day, in four equal doses administered orally at 8 a. m., 12 noon, 4 p. m., and 8 p. m. All determinations of blood sugar were made on samples of blood drawn before breakfast, while the patients were in a state of fasting.

The figures in the accompanying chart indicate the amount of blood sugar in milligrams per hundred mils of blood. The hatched areas represent periods of treatment with synthalin-B. In cases 2 and 4 the patients received a second course of treatment after the effects of the first course had disappeared, as indicated by the return of the blood sugar to a higher level.



Effect of Synthalin-B on Blood Sugar.—The hatched areas represent periods of treatment with synthalin-B. The figures indicate the number of milligrams of sugar in 100 mils of blood. The squares with heavy black outlines indicate days on which toxic effects were produced by the drug.

Three patients experienced toxic sequelae. This is indicated on the chart by the squares with heavy black outlines. Patient A. K. (case 2) complained of vomiting and abdominal cramps after having had the drug for four days. It is noteworthy that a second course of treatment of this patient, after she had rested for four days, was not accompanied by untoward effects. Patient E. S. (case 5) had nausea, vomiting and diarrhea after only two days of treatment (80 mg.). Patient O. C. (case 6) complained of headache and nausea but did not vomit. In all cases the symptoms vanished within twenty-four hours after the drug had been omitted. None of the patients showed any evidence of permanent injury. Icterus was not encountered.

An attempt was made to stabilize the level of the blood sugar by a uniform diet before synthalin-B was given. The reduction of the blood sugar shown in some of the cases before treatment with the drug was begun was due to the dietetic restrictions.

It will be seen that the blood sugar was lowered by synthalin-B in every instance, although in case 7 the extent of the depression was almost negligible. It appears, therefore, that synthalin-B is as effective as synthalin in lowering the level of blood sugar of patients suffering from uncomplicated diabetes mellitus. That it retains some of the toxic properties of its predecessor is shown by the symptoms experienced by some of the patients whose cases are reported here. I am now using slightly smaller amounts (30 mg. a day) with the expectation of avoiding some of the toxic effects.

That the drug does not affect all patients in the same degree, both as regards its beneficial and its deleterious action, is illustrated by the chart. For example, patient J. R. (case 7) took the drug for nearly six days without experiencing any ill results, whereas patient E. S. (case 5) was made sick after only two days of treatment. In case 7 a total of 230 mg. brought about an almost negligible reduction of blood sugar, whereas in case 5 a much greater lowering of the blood sugar resulted from giving only 80 mg. of the drug.

It is evident, also, that the effects of synthalin-B are prolonged beyond the time of treatment and may even become more pronounced. This is illustrated by case 4, in which the blood sugar was lower four days after the treatment was stopped than it was on the last day of treatment.

The mechanism by which the derivatives of guanidin reduce blood sugar is not known. It remains to be determined whether or not patients with diabetes are benefited in the long run by the use of these compounds. There can be no doubt as to their influence on the blood sugar, but this does not necessarily prove that the effect on the disease as a whole is favorable.

Synthalin-B can not yet be purchased in this country. Presumably it will become available later, but until further knowledge of its harm-

ful effects is acquired, it should be used with caution. It can not replace insulin in the treatment of diabetes mellitus, except possibly in some of the milder cases. In severe cases a supplementary rôle may be assigned to the drug to permit a reduction of the amount of insulin or, what I believe to be a more important consideration, a reduction of the number of hypodermic injections.

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TRICHINIASIS WITH WIDESPREAD INFESTATION OF MANY TISSUES

BY S. SEYMOUR HORLICK, M.D.,* AND RALPH E. BICKNELL, M.D.*

IN a review of the literature on trichiniasis, very few cases have been found in which extensive involvement of the whole body, particularly the central nervous system, has been observed. Since the discovery of the trichinae in the spinal fluid by Lintz and Van Cott in 1913, several clinicians, notably Salan and Schwartz, Meyer, Sterling and Hassin, have reported trichiniasis with symptoms referable to the central nervous system. No case report could be found in which there was simultaneous infestation of so many organs and tissues as in the following case which has come under the care of the writers.

REPORT OF CASE

Mrs. F. G., a white woman of 52 years, was admitted to the Lynn Hospital on August 12, 1928, complaining of severe headache, and swelling and pain in both eyes. Her family, marital and social histories were unimportant.

Past History: The patient was born in Canada, but had lived in Lynn, Massachusetts, for the past 25 years. She did not recall having been sick during childhood except for having had diphtheria twice. In 1918 she had influenza. She was never operated upon and stated she had always been healthy. The patient was injured in an automobile accident two years ago, sustaining fractures of the left zygoma and left temporal bones. Following this, she had occasional headaches over the left temporal region, which were relieved by aspirin. These headaches were not so severe as to interfere with her work. *Eyes:* Vision impaired—wore glasses; no inflammation or pain. *Ears:* Hearing somewhat impaired for past few years. No discharge or pain. *Nose:* Epistaxis frequent before climacteric—none since then. No obstruction; not susceptible to colds. *Teeth:* Upper teeth false; lower teeth caused her occasional toothache. *Throat:* Had numerous sore throats, especially in spring and autumn. Tonsils and adenoids not removed.

Gastro-Intestinal: Appetite always good; ate regularly; not finicky about food. Bowels moved daily with occasional catharsis. No gas, colic, icterus or diarrhea. Prior to her climacteric the patient had hematemesis, which started on the first day of her menstrual period and continued once or twice each day for five days. This hematemesis recurred every twenty-one days, coincident with slight vaginal staining from the onset of the menses at twelve years to the climacteric at forty-five years. There was no history of hemorrhoids, or of tarry, bloody or clay-colored stools. She had no history of symptoms referable to her cardio-respiratory, genito-urinary or neuro-muscular systems. Weight stationary at 199 pounds for many years.

Present Illness: The patient was taken suddenly ill with a severe headache on August 9, 1928, three days prior to her admission to the hospital. This ache started at the back of the neck and radiated upward and forward to the occipital, parietal and frontal regions. It was constant and so severe as to interfere with her sleep. The following day she noticed both her eyes were closed by edema, which was accompanied by orbital pain. This edema and pain persisted for about thirty-six hours, after which both disappeared. The headache persisted. At this time, she began to feel "soreness" of both shoulders and the back of her neck, which was intensified and made excruciating by moving her head. The patient then called her family physician, who advised hospitalization.

On August 1, 1928, eight days before the appearance of severe symptoms, the patient had for lunch a broiled pork chop "well done and browned". This was the first time in many weeks that she had eaten meat. Three days later, she took some Epsom salts because she "felt out of sorts". This was followed by profuse watery stools, five or six per day, for three days.

The patient's daughter had eaten some of the pork chops at the same meal, but complained of no symptoms except a "cold in the back", which lasted for two days, August 7th and 8th, and was characterized by pain and soreness in her back.

Physical Examination revealed an obese white woman, lying flat in bed, very uncomfortable, with severe headache. Her face presented an anxious expression,—flushed and moist with perspiration. Her temperature was 103°F. She was conscious, rational and co-öperative. *Skull:* Symmetrical; no exostoses, irregularities or points of tenderness. *Scalp:* Clean, with slight scarring over left temporal region. *Hair:* Brown-gray, soft and abundant. *Face:* Anxious expression; perspiring. Pitting edema over both zygomas. *Skin:* Hot and moist; no eruptions or scaling. *Eyes:* Pupils equal and regular. They reacted to light and accommodated to distance. No nystagmus or gross disturbance in vision. There was evident edema of the eyelids with injection of the lower palpebral conjunctivae. No strabismus. *Ears:* Hearing fair; no discharge or tophi. *Nose:* No deformities or discharge. *Mouth:* Fetid breath. No ulceration or pigmentations. *Teeth:* Wore partial upper plate. Lower teeth showed many caries. *Tongue:* Heavy white furring. Protruded in midline without tremors. *Pharynx:* No congestion. Tonsils were atrophic. *Neck:* Muscles of back of neck exquisitely tender. Thyroid not palpable. No abnormal pulsations. No palpable lymph nodes. *Thorax:* Symmetrical; expansion equal on both sides; no abnormal pulsations. *Breasts:* Pendulous; no scars or palpable masses. *Heart:* Apex beat not seen but felt in fifth left intercostal space, 10 cm. from mid-sternal line. Left border of cardiac dullness corresponded to apex beat. No murmurs heard.

*For record and address of author see "This Week's Issue," page 843.

$A_2 > P_2$. Heart sounds forceful and of good quality. No pulse deficit. *Peripheral vessels:* Palpable but compressible. Blood pressure not determined because of pain in both arms. Pulse rate 118. *Lungs:* Clear and resonant throughout. No abnormal breath sounds or râles. Respiratory rate 18. *Abdomen:* obese but symmetrical. There was some superficial tenderness over the left side of the abdomen. There were no palpable masses. There was spasm of the muscles over the right upper quadrant and questionable palpable liver border about 2 cm. below costal margin. Spleen and kidneys were not felt. *Extremities:* There were several points of tenderness in the biceps and triceps of both arms. There were other points of tenderness diffusely scattered through the muscles of shoulders, forearms and back. *Reflexes:* Normal knee jerks. No ankle clonus or Babinski reflexes. No Kernig. Brudzinski reflex not elicited because of pain induced by moving the head. *Sensations:* Not impaired.

The urine on admission was amber colored, with a specific gravity of 1.026. It was acid in reaction and showed a slightest possible trace of albumin but no acetone or sugar. The sediment contained abundant squamous epithelium from the bladder and 20-30 leucocytes per high power field.

The red blood corpuscles numbered 5,000,000 and white cells 8,950. The hemoglobin was 85%. Differential count of 300 cells showed polymorphonuclear neutrophiles 69%, polmorphonuclear eosinophiles 23% and lymphocytes 8%. Centrifugation of the whole blood after treating with 10 volumes of 3% acetic acid showed the presence of the larval form of Trichina spiralis.

A diagnosis of trichiniasis was made evident by the characteristic clinical picture of edema of the eyelids, muscle pain and tenderness, and eosinophilia. This diagnosis was confirmed by finding the larval form of the Trichina spiralis in the blood and later by biopsy on sections of muscle tissue.

Muscular pain and tenderness was constant and severe. Opiates gave no relief. Eight days after admission to the hospital, there was a temporary relief of headache, which had been a prominent symptom. Coincident with this relief there was a drop in temperature and pulse by lysis to normal. Physical examination at this time revealed a hyperactivity of knee jerks, biceps, triceps and Achilles reflexes. There was present a bilateral Babinski reflex but no ankle clonus. The patient appeared stuporous. Her lungs were clear and resonant though her respirations were rapid and labored. The liver edge was palpable 4 cm. below the costal margin. Eosinophilia now 43%.

The following day fixation of the eyeballs was noted. Movement of the eyes in any direction provoked pain. There was now definite incoherence of speech and disorientation. The patient showed spastic paralysis of the left arm and leg.

These symptoms to us were significant of new infestation of the central nervous system, the diaphragm and the extrinsic muscles of the eye. We did not understand the enlargement of the liver, at this time, but it was made clear at autopsy (vide *infra*). The patient was seen in neurological consultation by Dr. H. R. Viets, who made a diagnosis of toxic psychosis and encephalitis,—both secondary to infestation of muscles.

During the next few days, the patient showed signs of improvement. She was mentally more alert. The pain and tenderness had gradually diminished. The eosinophilia was reduced to 6%.

On September 1, twenty-three days after admission, our patient again had a relapse. She became stuporous; her temperature and pulse began to rise; tenderness and pain in the muscles became more diffuse and severe. The left border of cardiac dullness measured 15 cm. from the mid-sternal line. No murmurs were audible. The heart sounds were of fair quality, but were extremely rapid (150 per minute). The following day the lungs showed dullness at both bases, with coarse moist râles. Her respirations were extremely rapid and labored (60 per minute). Her temperature stayed about 104 F. by axilla for two days, then rose to 106.2 F. on September 4; she became moribund and died.

This last relapse was probably due to infestation of the heart muscle by the Trichina spiralis, with a resultant hypostatic pneumonia.

Permission was obtained to do a post-mortem examination through a limited abdominal incision. The following is the report by Dr. J. S. Rooney of his findings at autopsy. (Because of the unusual findings it is given in detail.)

Post-Mortem Report

BODY: Is that of a well developed, very obese, white female measuring 176 cm. There is practically no rigor present. There is slight post mortem settling of blood in the dependent portions and right side of face. The external ears and nasal passages are clean. The eyes are blue, the cornea soft and steamy. The pupils are blue, are equal, and measure 5 mm. in diameter. There is some sordes about the base of the teeth. There is a bed sore over the sacrum. There are many small petechial hemorrhages scattered over the body.

PRIMARY INCISION: The incision is limited to the abdomen. The fat over the abdomen measures about 6 cm.

PERITONEAL CAVITY: The omentum contains a large amount of fat. There is no free fluid in the peritoneal cavity. The appendix is retrocecal and appears normal.

PLEURAL CAVITIES: There is no free fluid in either cavity. There are old fibrous adhesions between the lower lobe and the anterior chest wall.

PERICARDIAL CAVITY: The heart was removed from below and nothing abnormal noticed about the pericardium or the pericardial sac.

HEART: Weighs 365 gms. The T.V. measures 11½ cm.; P.V. had been cut in removing the heart; M.V. 10 cm.; A.V. 7 cm. The left myocardium measures 1.6 cm. and the right 4 mm. The valves appear normal. The musculature of the heart is softer than normal and brownish in color. Under the pericardium is considerable fat.

Sections microscopically through the heart muscle reveal the presence of several areas of necrosis, and the contents of these necrotic areas are missing. There is marked cellular infiltration around the missing tissue, many of these cells being eosinophils. An occasional area is seen in which an organism characteristic of trichina can be made out. There is some increase in the fibrous tissue of the heart and this tissue shows moderate round cell infiltration. The coronary vessels are slightly thickened, but are patent. The endocardium and myocardium are normal in appearance.

LUNGS: The right lung weighs 555 gms.; left 455 gms. There is settling of blood and edema in the posterior portions of the lung. The anterior portion is air containing.

Sections microscopically through the lung tissue show the presence of marked edema and congestion. There is no evidence of any infectious process and no trichinae are seen. No areas of necrosis are noted. There is some anthracosis scattered throughout the lung tissue, particularly just beneath the pleural surface. The pleura is slightly thickened.

LIVER: Weighs 1960 gms. It is softer than normal and shows considerable fatty change about the veins. The liver is of the nutmeg type. There is some bile stasis in the liver.

Sections microscopically through the liver show the tissue to be markedly congested and the sinusoids filled with red cells. In some areas there is marked brown atrophy of the liver cells, and the

cells are markedly pigmented. There is some fatty infiltration. Careful search fails to reveal the presence of any areas of necrosis or any parasites. The capsule does not appear thickened.

GALL BLADDER AND DUCTS: The bile ducts are patent. The gall bladder is markedly distended and very pale. It is filled with a clear fluid and contains many small, pale, straw-colored stones.

PANCREAS: The pancreas is rather soft due to a fatty ingrowth in the lobular stroma.

Sections microscopically show marked infiltration with fat, but aside from this are essentially normal in their appearance. The islands of Langerhans are very prominent. An occasional area, which stains intensely and has the characteristic appearance of a parasite as seen in the muscles, is made out.

LABORATORY DATA

BLOOD

	Date	W.B.C.	R.B.C.	Hgb.	Lymph.	L.M.	P.N.	P.E.	P.B.	Remarks
	Aug. 12	8,950	5,000,000	85%	8%		69	23		Embryos in blood
A.M.	" 13	20,100		80%	13	1	68	17	1	No embryos seen
P.M.	" 13	20,800			13	1	55	31		" " "
	" 16	20,400			20		37	43		" " "
	" 18	20,600			7	6	60	27		" " "
	" 20	22,700			7	1	64	28		" " "
	" 22	27,400			17	3	57	23		" " "
	" 23	18,800			11		71	18		" " "
	" 24	18,000			17	1	66	15	1	" " "
	" 25	17,900			15	1	75	9		" " "
	" 28	20,600			22		72	6		" " "
	" 30	16,250			27		59	13	1	" " "
	" 31	19,850			13		77	10		" " "
	Sept. 1	27,800			17	3	73	7		" " "

URINE

	Sp. Gr.	React.	Alb.	Acet.	Sugar	Sediment
Aug. 13-'28	1,026	Acid	Spt.	Neg.	Neg.	Much squamous epith. 20-30 wbc/hpf.
Aug. 22-'28	1,024	Alk.	Spt.	Neg.	Neg.	Frequent squamous epith.; Occas. triple phosphate; Occas. wbc.
Aug. 29-'28	1,027	Alk.	Vst.	Neg.	Neg.	Occas. triple phosphate Amorphous urates.

OTHER LABORATORY DATA

Aug. 15-'28	Wassermann negative
Aug. 22-'28	Widal negative
Aug. 14-'28	No parasites in stool
Aug. 16-'28	" " " "
Aug. 17-'28	" " " "

Aug. 22-'28 Biopsy - reported - Trichiniasis.

Post Mortem:-

Spinal fluid and gall bladder fluid centrifuged, - both show presence of parasites.

SPLEEN: Weighs 300 gms. It is very soft and shapeless. It flattens out when laid on the table. The spleen is a dull purplish-blue color. On cut surface the pulp is very soft and mushy. The pulp scrapes off very easily with a knife. The spleen is of the septic type.

Sections microscopically show the spleen to be composed almost entirely of red cells. Only an occasional splenic corpuscle is made out. The capsule is not thickened. No areas of infection are noted and no parasites are seen. The tissue has the characteristic appearance of a toxic or septic spleen.

KIDNEYS: The right kidney weighs 225 gms.; the left 215 gms. The cortex measures 7 mm. The capsule is somewhat adherent; when stripped off leaves a granular roughened surface. The cortex is pale and shows yellow streaks. The pyramids are pale. Several small pinpoint yellowish areas are seen in the pyramids. There is injury to the cortex of the kidney.

The kidneys are remarkable in their appearance in that numerous parasites are seen scattered throughout the tissue. Around the parasites is an area of necrosis and there is marked cellular infiltration

with polymorphonuclear cells. The predominating type of cell is the eosinophil. In many places, due to the softening of the tissue, the parasite has fallen away and has left a small hole. There is considerable hemorrhage between the tubules and some increase in fibrous tissue. The glomeruli are normal in appearance. The blood vessels show some thickening characteristic of arteriosclerosis. The capsule is slightly thickened.

BLADDER: The bladder is well contracted down. The mucosa has a little exudate on the surface near the urethral opening. There is some hemorrhage into the mucosa. The bladder shows a cystitis.

GASTRO INTESTINAL TRACT: In the small intestines, small slightly raised reddish areas were seen in the mucosa. Sections will be made of these areas.

Sections microscopically reveal the presence of numerous characteristic parasites scattered throughout the mucosa, submucosa and muscularis. There is an area of necrosis about each parasite. The cellular reaction in and around the organism is mostly of the eosinophilic type.

MUSCLE: Small pieces of tissue are taken from the diaphragm, pectoral, abdominal and thigh muscles. In the abdominal muscles small pinpoint, yellowish white areas are seen. Grossly it is not possible to say they are trichinae.

Sections taken from the diaphragm, abdominal muscles, psoas muscle, and thigh muscles all show areas of necrosis, such as have been described in the other tissue. Trichinae are seen scattered throughout all of the sections. Sections microscopically through the tip of the tongue show the characteristic reaction as described in the other musculature.

Gall bladder fluid and spinal fluid obtained at autopsy showed the presence of the larval form of the Trichina spiralis.

COMMENT

This is an unusual case in that so many varied organs and tissues were involved. Of interest, particularly, was the finding of the encysted trichinae in the heart muscle, pancreatic tissue, kidneys and intestinal musculature. It is to be regretted that permission to examine the brain and spinal cord could not be obtained.

What we thought to be liver proved at autopsy to be an enormously distended gall bladder filled with straw-colored fluid, from which trichinae were recovered.

The method of Staubi was followed in demonstrating the parasite in blood, spinal fluid and gall bladder fluid. His method consists in mixing several cubic centimeters of the suspected fluid with ten times this quantity of 3% acetic acid and then treating the centrifuged sediment as a blood preparation.

LEAD POISONING

It has recently been stated by Surgeon General H. S. Cumming of the U. S. Public Health Service that lead poisoning is one of the great problems of industrial hygiene. In the early part of the 19th century, experimental investigations of the action of lead were begun; but it is only during the past 25 years that much progress has been made in methods of preventing lead poisoning. The application of knowledge of how to prevent it, together with improved methods in the handling of poisonous materials, will greatly reduce the incidence of this and other industrial poisonings in the future.

The following are some of the principal industries and trades in which workers are exposed to lead: Lead mining and smelting, making white or red lead; the manufacture of paint, storage batteries, glazes, enamels, pottery, and most lead objects; painting, brass founding, and polishing cut glass and brass. In general all of those who handle lead-containing materials, even such diverse things as putty and window-shades, are exposed more or less to lead poisoning. The State of Maine has had a number of deaths from lead poisoning which were not at all industrial in the ordinary sense of the word. These cases of poisoning were among the people who resided in rural communities and used lead piping to bring spring water to their houses. The lead from the pipes was dissolved and poisoning resulted from using this water. This condition has also been found in many other States.

When a person is exposed to a dangerous amount of lead, he begins to absorb it and his system will show evidence of it very early. There are many people who absorb lead and show its disabling effects and yet do not have acute poisoning. If the amount of lead is insufficient to produce acute poisoning and the worker continues in the occupation offering exposure, he may develop lead poisoning in a chronic form, which is at times harder to overcome than the acute condition.

Symptoms of a disease are those things of which the patient makes a complaint. To him, they are paramount; but the physician may find that there are more serious damages done to other parts of the body, about which no complaint has been made. This is true in lead poisoning. Intestinal cramps are quite prominent and are often the first and most conspicuous symptom that the patient notices; yet, lead damages other organs and tissues far more than it does the intestine. The blood is one of the first tissues to show changes when lead is taken into the body, and these changes may be very marked in cases of heavy exposure.

One of the most painful symptoms of lead poisoning is what is commonly called "lead colic". The action of lead on the intestine produces severe cramping pains. These are usually accompanied by another common sympton, obstinate constipation. This has been attributed to the action of lead on the musculature of the intestines.

Lead is eliminated from the body principally by the kidneys and intestines. A very important thing to remember in exposure to lead is its storage in the bones. While the lead is stored in the bones, it does not produce any harm, apparently. The body, however, makes an effort to relieve itself of this poison, and as a consequence, small amounts of it are eliminated long after exposure to lead has ceased. This elimination can be hastened by the ingestion of certain substances which bring about chemical changes in the body. Also, illness and excesses, such as alcoholism, bring about chemical changes which increase the rate of elimination of lead from the bones. When it is thus free again in the circulation, it may produce evidence of its poisonous effects as in the beginning; cases of acute poisoning have been recorded in such instances.

Persons who have been exposed to lead should avoid constipation and otherwise aid elimination in every way. The saline purgatives are of particular value in removing lead; fruits are also very de-

(Continued on Page 832)

MEDICAL PROGRESS

PROGRESS IN TUBERCULOSIS, 1928-1929

BY JOHN B. HAWES, 2ND, M.D.,* AND MOSES J. STONE, M.D.*

DIAGNOSIS

AUSTRIAN, CHARLES R. (*Am. Rev. Tub.*, July, 1928, XVIII-37) makes a plea for more carefully collated history and more painstaking physical examination of the patient in the examination for a question of pulmonary tuberculosis. He contends that bacteriological laboratories furnish valuable data, but are only accessory diagnostic criteria, and are often lacking in very early cases.

Hawes, John B. 2nd, (*N. E. J. M.*, April 5, 1928, CXCVIII-336) discussing the subject of early diagnosis of pulmonary tuberculosis, finds that the method of attack must be changed if we expect to make early diagnosis. He deprecates the fact that the entire subject of tuberculosis is given too scanty attention in our medical schools, also that most general hospitals refuse to admit tuberculous patients. He feels that while blood-spitting, pleurisy, and persistent cough should be prominently mentioned, the constitutional signs and symptoms such as chronic fatigue, undue fatigue, ease of tire, should be greatly more stressed.

Molony, I. C., (*Am. Rev. Tub.*, Jan. 1929, XIX-99) studying the significance and diagnostic value as well as the frequency of certain signs and symptoms in pulmonary tuberculosis, found that a large proportion of people have such a high tolerance for tuberculosis that they will carry it even to an advanced stage before breaking down. He found the average interval between the first-known symptoms of onset and the time of breakdown to be two years and three months. The number of patients who volunteered no complaints referable to the chest on admission was large enough to emphasize the fact that failure to complain of chest symptoms does not rule out pulmonary tuberculosis. 85.7% of patients drew attention to the lungs. Abdominal disturbances constituted the fourth most common complaint on admission. He found pain in the chest to be the most prominent and reliable symptom. It was the third most frequent complaint on admission and the second most frequent under treatment. It occurred twice as frequently as chills and sweating. Over 60% had no definite history of tuberculosis in the immediate family. Positive sputum was found only in 60.8% of the cases.

Clifford, R., (*N. E. J. M.*, June 21, 1928, CXCVIII-948) studying the significance of signs in pulmonary tuberculosis, found that symptoms are more important than physical signs in judging activity in pulmonary tuberculosis. He found that fine apical râles may persist for a long time in cases of healed tuberculosis. He believes that such râles do not necessarily depend on moisture in the bronchi secondary to inflammatory reaction, but may be due to collapse of lung lobules. He feels that such râles in the absence of symptoms do not warrant a diagnosis of active pulmonary tuberculosis.

(Clifford's conclusions must be confirmed before being accepted.)

Petschacher, L., (*Wien. Klin. Wchnschr.*, July 5, 1928, XLI-964) found that most frequent diseases erroneously diagnosed tuberculosis are influenza, anaemia, chlorosis, heart disease, and cancer; less frequently also in chronic pneumonia, chronic sepsis, pulmonary tumors, chronic pulmonary abscesses, bronchiectasis, and thoracic lymphogranulomatosis.

(Chlorosis and lymphogranulomatosis are too rare to merit much consideration.)

Weiss, H. B., (*J. A. M. A.*, Jan. 21, 1928, XC-187), finds that lateral roentgenographic views may be sometimes of great value, especially when the lesion is behind or in the front of the heart. He does not, however, recommend it in early tuberculous involvement as the superposition of the lungs easily obscures the finer changes.

Pottenger, F. M. (*Am. J. M. Sc.*, May, 1928, CLXXV-674) found that when small lesions are suspected on physical examination the patient should be x-rayed dorsoventrally if the lesion is suspected anteriorly, and ventrodorsally when suspected posteriorly, or, what is better, plates should be taken in both directions.

(This is rather a fine point.)

Hayek, H. V. (*Wien. Klin. Wchnschr.*, June 21, 1928, XLI-891) finds that it is a frequent and grave mistake to over-emphasize the obvious and dense shadows of old cirrhotic lesions on roentgenological examinations, and to overlook the soft shadows of fresh processes. He recommends repeated films in order to interpret new developments. He feels that an early diagnosis of cavitation is impossible without x-ray studies; the same is true of early infraclavicular infiltrations.

Hawes, John B. 2nd, (*Boston M. & S. J.*, Nov. 24, 1927, CXCVII-961) studying the relationship of upper respiratory infection to pulmonary tuberculosis, finds that a certain number of symptoms related to the lungs, such as coughing, expectoration, etc., and another group of symptoms strongly suggesting pulmonary tuberculosis, such as loss of weight and strength, rapid pulse and fever, may be due to infections of the upper respiratory tract. He strongly

*For record and address of author see "This Week's Issue," page 843.

urges that this possibility should always be borne in mind. He feels that the co-öperation between the internist and rhinologist is very important.

Ulrici, H. (*Klin. Wchnschr.*, May 27, 1928, VII-1045) found the super-refined diagnosis of incipient apical tuberculosis has resulted in the admission to German sanatoria of as many as 80% of patients with an allegedly incipient and often imaginary apical focus. He feels that only about 7% of the so-called apical lesions develop into progressive processes. He recommends that only patients with infraclavicular (incipient foci or with acute recurrences) should be admitted to the sanatoria.

(This is a radical and likewise crude criterion on which to decide so important a point.)

PROGNOSIS

Barnes, Harry L., and Barnes, L. R. P., (*Am. Rev. Tub.*, Oct. 1928, XVIII-412) making a statistical study of the duration of life in phthisis with cavity, found that 80% of the patients died within one year; 90% were dead within five years. The average duration of life of 270 cavity cases from the appearance of the cavity signs to death was 15.8 months. Ninety-nine per cent. of 616 cases diagnosed by x-ray had tubercle bacilli in the sputum. Only one of 57 colored patients survived three years. They also found that the percentage of one- and three-year survivors was in direct relation to the amount of pulmonary disease. The duration of life also bore a direct relation to the number of cavities. Of 17 cases having more than three cavities none survived a year. Eighty-eight per cent. of patients having cavities larger than 7 c.m. died within a year.

Pinner, M., and Werner, W. I., (*Am. Rev. Tub.*, Oct. 1928, XVIII-490) in a study of more than 500 adult patients with active pulmonary tuberculosis, found tubercle bacilli in the sputum in over 99%. They also found that the absence of tubercle bacilli in the sputum of patients with pulmonary tuberculosis almost always indicates healing. They feel that negative sputum has a much greater diagnostic and prognostic significance than represented by the usual textbook teaching. They urge all sanatoria to invest in facilities for the complete examination of sputa from all patients.

Levinson, A. (*Am. Rev. Tub.*, May 1929, XIX-499), in a study of 119 cases of active tuberculosis, mainly in negro children, found pulmonary involvement in 99 cases, the other 20 cases having suffered from tuberculosis in other parts of the body. Sixty-nine were miliary; 15 acute tuberculous pneumonia and 15 subacute or chronic pulmonary tuberculosis. He found the tuberculin test negative in all cases of miliary tuberculosis. The prognosis in all the acute cases was bad. Of the 15 chronic cases only one is doing well. He believes that the high mortality is due to the fact that the disease is far advanced when the patients come to the hospital.

TREATMENT

Jacobeaus, H. C. (*Am. Rev. Tub.*, July 1928, XVIII-94) states that the purpose of all surgical treatment in pulmonary tuberculosis is to bring about collapse of the diseased organ either in its entirety or in part. In the main he recognizes three forms of surgical procedure:

(1) Treatment by artificial pneumothorax, mainly elaborated by Forlanini and Saugman, including cauterization.

(2) The method of thoracoplasty elaborated by Sauerbruch, Brauer and others.

(3) Phrenicotomy for therapeutic purpose first suggested by Sturtz. He advises very strongly to consider pneumothorax before thoracoplasty is resorted to. The author also outlines the indications and methods for cauterization of adhesions in cases of incomplete pneumothorax. He also feels that thoracoplasty is feared too much and that it ought to be used more than is the case.

Whittemore, W., (*Boston M. S. J.*, Jan. 6, 1927, CXCVI-1) discussing the different surgical procedures in the treatment of pulmonary tuberculosis, feels that the results of surgical treatment are amazing when one considers the poor conditions of the patients. Although in his series of cases there was 97% mortality, others have reported only 4-5%.

Miller, J. A., (*Am. Rev. Tub.*, Nov. 1928, XVIII-523) discussing climate as a factor in the treatment of pulmonary tuberculosis, finds that the regimen of regulated rest and exercise, proper food, and open-air life is the fundamental essential in the treatment of tuberculosis. Suitable climatic environment makes this open air life more easy, enjoyable and beneficial. He concludes that there is no universally ideal climate. For each patient there may well be a most favorable environment if we are fortunate enough to find it. He recommends reasonably cold, dry, variable climate, such as is found in the mountains, for young or vigorous constitutions which will react well, dry, sunny climates for laryngeal cases and those with marked catarrhal secretion, equable, mild climates at low altitudes for the elderly and those of nervous temperament as well as those with arteriosclerosis, weak hearts, or marked tendency to dyspnoea.

Fischel, Karl (*Am. Rev. Tub.*, Nov. 1928, XVIII-551) makes a plea for a better selection of cases for thoracoplasty operation. He deplores the fact that the operation has been used as a last resort in too many hopeless cases. On the other hand, suitable cases that could be saved are frequently not subjected to surgery. He feels that better results will be attained if the selection of cases is being improved, not only by excluding unsuitable patients from thoracoplasty, but also by making better and

more frequent use of this valuable operation in proper cases. The selection of cases has to take into account not only the pathologico-anatomical nature of the tuberculous lesion but also the physiology and pathology of respiration and circulation.

Pottenger, F. M., (*Am. Rev. Tub.*, Nov. 1928, XVIII-570) discussing what is meant by clinical tuberculosis, answers that it is more or less active tuberculosis which is indicative of a continuous absorption of toxins. He feels that minor stimulations result in increased immunity and healing. Severe re-inoculations, if the patient is able to overcome them, are followed by very high immunity and healing. If the patient fails to overcome them, immunity is depressed and the disease spreads. He advocates the therapeutic use of tuberculin injections, thus following nature's method of anti-tuberculin treatment.

(Very few will agree with Pottenger in advocating the use of tuberculin in lung cases.)

Kendall, W. B., and Ross, C. B., (*Am. Rev. Tub.*, Dec. 1928, XVIII-804) making a comparative study of immediate and late results of treatment by artificial pneumothorax, find that the immediate results are confirmatory of those satisfactory results previously found. Late results as found at the end of ten years are satisfactory when compared with the results shown by cases in which compression was not established. They found that the temporary good results are found predominantly in the cases in which pulmonary compression is not complete, and the benefits so achieved are greatly diminished at the end of the first five-year period. They further state that the presence of a definite lesion in the contra-lateral lung does not prohibit the use of uni-lateral pneumothorax provided that care is used in estimating the extent and activity of this lesion.

Peters, Leroy S. (*Am. Rev. Tub.*, Jan. 1929, XIX-74) advises bed-rest for tuberculous patients before any other procedure is contemplated. He advises against use of external appliances such as pneumatic splints, hard pillows, etc., for an indefinite period of time in lieu of the more effective forms of compression. He found phrenicotomy to be effective only in basal lesions and as a preliminary to thoracoplasty. He reiterates again his previous statements that artificial pneumothorax is the most efficient of all forms of compression when a good collapse is possible. He advises thoracoplasty only when the collapse is partial and cannot be made effective by cauterization after the method of Jacobeaus. He concludes that thoracoplasty offers a better end-result than a partial pneumothorax, but does not compare favorably with a complete pneumothorax.

Stoll, H. F. (*Boston M. & S. J.*, Dec. 1, 1927, CXCVII-1017) found the common mistakes made in the management of a tuberculous patient are: (1) failure to appreciate and instruct the patient in regard to absolute rest; (2) overfeeding; (3) failure to teach the patient about his disease; (4) failure to recognize symptoms suggesting intestinal tuberculosis; (5) neglect of details; lack of specificity; (6) neglect of operative procedures such as pneumothorax, phrenicotomy or thoracoplasty.

Burrell, L. S. T. (*Brit. J. Tub.*, Jan. 1928, XXII-13) found that spontaneous pneumothorax of tuberculous origin usually goes on to the formation of effusion. In some cases this clears up after one or more aspirations, but, usually, it leads to a chronic pyopneumothorax. In this case, if the lung cannot re-ëxpand early, he advises thoracoplasty. He also found that in some cases spontaneous pneumothorax is beneficial and it is best to prevent the lung from re-ëxpansion by keeping up artificial pneumothorax.

Varrier-Jones (*Brit. M. J.*, Dec. 31, 1927, No. 3495) believes that the village settlement is the ideal scheme for the after-care of the tuberculous.

Gexler, W. A., and Weigel, B. J. (*J. A. M. A.*, Feb. 25, 1928, XC-590) report the favorable results they obtained by postural rest in pulmonary tuberculosis. Their method is to have the patient lie on the affected side on a pillow which is especially made for this purpose. The thickness is three inches, width, 10 inches, and length, 20 inches. The patient begins with one hour of postural rest the first day, increasing a half-hour daily to a maximum of 16-20 hours. They report a striking amount of healing in one-fourth of the total patients treated.

Liebermeister, G. (*Beitr. z. Klin. Tb.*, 1927, LXV-567) recommends the use of postural drainage in pulmonary tuberculosis. He also advocates its use as an adjunct to collapse therapy.

Berard, L. and Deumarest, F., (*Rev. de la Tuberc.*, April 1927, VIII-222) reviewing the results they obtained by surgical treatment of pulmonary tuberculosis, feel that surgery has more than justified itself. Of course, the results obtained will be improved by perfection of technique and selection of material. They express their confidence in the great future that surgery has in phthisiotherapy.

Whittemore, W. (*Boston M. & S. J.*, Jan. 26, 1928, CXCVII-1395) believes that thoracoplasty should always be done in two or more stages. He does not favor phrenicotomy, believing it of little additional value if the main operation is complete. He feels that the results of surgery are striking if one considers the poor condition of the patients previously. Quoting Alexander, he states that one may expect one-third to be cured with no bacilli in the sputum and be able to work, one-third to be definitely improved, and the remaining third to be unimproved.

Mattison, S. J. and Hoit, H. A., (*Am. J. Surg.*, March 1928, N. S. IV-305) argue that surgical collapse should be instituted from a physiological rather than a pathological consideration, as

atelectasis is the normal state of the lung at rest, and is present about foci of disease, separating disease from healthy tissue. He believes that it should be done earlier than it is at present, both because of the damage it may prevent and the good it may do after damage has been done. He feels that other advantages from surgical treatment of pulmonary tuberculosis are that it saves the patient time and money, does away with the necessary close co-öperation between patient and physician, and anticipates the crippling effects of natural healing.

Matson, Ralph C. (*Am. Rev. Tub.*, March 1929, XIX-233) reports on cauterization of adhesions according to the method of Jacobeaus utilizing Unverricht's thoracoscope. He modified the procedure by using the endothermic method rather than galvano-cauterization. He feels that the technique of cauterization of adhesions by the Jacobeaus-Unverricht method is simple and free from danger. He states that he is convinced of the value of the Jacobeaus-Unverricht operation, and regards it as a distinct contribution to collapse therapy. He found the operation of cauterization to be a minor affair to the patient, while, if successful, it may convert a useless pneumothorax into a satisfactory one and save the patient from thoracoplasty. He believes that the operator should have experience in phthisiology and surgery and that the use of the method should be confined to physicians with a pneumothorax background and experience in tuberculosis or to phthisiologists who have had some surgical training.

Berard, Guilleminet and Desjacques, (*Rev. Belge D. L. Tuberc.*, March-April, 1927, XVIII-49) find phrenicotomy a supplementary recourse in collapse therapy not to be underestimated and neglected. They urge its use as a supplement in partial artificial pneumothorax; also, combined with thoracoplasty, it diminishes the operative risk. In general, they advise phrenicotomy in fibrocaseous or progressive tuberculosis after failure of pneumothorax and when thoracoplasty is not applicable.

Moreau, J. and Olbrechts, E., (*Rev. Belge D. L. Tuberc.*, March-May 1926, XVIII-123) feel that the ideal theoretical indication for phrenicotomy is a lesion limited to the base. In such a case phrenic exeresis is preferable to pneumothorax. Other indications are: (1) impossibility, because of pleural adhesions, of effecting a pneumothorax; (2) as a substitute for thoracoplasty in patients in poor condition or unwilling to have major surgery; (3) in preference to pneumothorax in certain unilateral lesions. Contraindications are exclusively or essentially apical lesions and active bilateral lesions.

Matz, P. B. (*Am. J. M. Sc.*, July 1928, CLXXVI-87) reviewing the results of artificial pneumothorax treatment in the U. S. Veterans Bureau hospitals, found that 2.5% of the total number of tuberculous patients had pneumothorax treatment. He states that the best results with pneumothorax therapy were obtained in patients with bilateral lesions who had less than 1/3 of the better lung involved; the next best results were obtained in patients with unilateral involvements; the poorest results were obtained in patients with bilateral lesions who had more than 1/3 of the better lung involved. He found that there was a decrease of the lesions in the noncompressed lung according to physical signs in a little less than 1/2 of the patients.

HILUM TUBERCULOSIS

Fraser, F. H. (*Am. Rev. Tub.*, Sept. 1928, XVIII-336), studying the co-existence of tuberculous cervical and mesenteric lymph nodes as well as tracheo-bronchial lesions by means of roentgenographic examinations, found that out of 151 children, including 31 with known tracheo-bronchial lesions, only two cases had cervical calcium deposits. Four cases had cervical tuberculosis without demonstrable calcium. Abdominal calcified nodes were present in two cases without signs or symptoms. All roentgenographically positive cases reacted positively to tuberculosis and were without pulmonary lesions.

Chadwick, H. D. (*Boston Med. & Surg. J.*, Jan. 26, 1928, CXCVII-1399) estimates the incidence of hilum involvement in school children to be from nine to ten per cent.

Wahneau (*Beitr. z. Klin. D. Tuberc.*, 1927, LXVII-601) recommends three aids for determining hilum tuberculosis in children: a good history, testing with tuberculin, and roentgenography. He feels that active bronchial lymph node tuberculosis is a rarer disease than it is generally considered. He warns against making a definite diagnosis on a single examination.

Wasson, W. W. (*Boston Med. & Surg. J.*, Feb. 9, 1928, CXCVII-1493) studying the differential diagnosis of hilum infection found, in tuberculous infection, the lymph nodes to be discrete with some evidence of congestion, fibrosis or calcification, also radiating lines from the hilum, beaded in appearance. Clinically, the patients are frequently underweight, show a slight and variable temperature increase, and in the adult malaise, in the child nervousness. He found that the typical non-tuberculous case does not show the discrete nodes but rather an evidence of connective tissue increase, especially about the radix. The patients are also more apt to be well nourished or overweight. He feels that a radiograph with considerable connective tissue about the radix pulmonis, without much enlargement of the outer broncho-pulmonary lymph nodes, and without radiating lines extending into the interior, looks like an upper respiratory infection, especially if there is sinus or tonsillar infection.

(His conclusions, based on such slender and meticulous evidence, cannot be accepted.)

Miller, R. H. and Shedden, W. M., (*Am. Rev.*

Tub., May 1929, XIX-511) feel that in a considerable number of cases of early tuberculous cervical adenitis, whether in children or adults, and often in cases of longer duration in which operation is technically feasible, radical surgery is the method of choice. The operation must be supplemented with all the supportive and palliative measures which may be used.

Bronfin, L. D. (*Am. J. Dis. Child.*, Nov. 1928, XXXVI-931) found the greatest incidence of clinical disease and positive physical signs in children with a history of definite exposure. Physical signs were often negative when the history and symptoms pointed to active disease. He noted fever and fatigue in about 50% of known contact cases. X-ray examinations showed normal lung in 24%, 86% showed enlarged hila, 65% manifested calcification in the hilar regions, 22% showed tracheo-bronchial lymph node enlargement, 11.6% showed primary foci and 11.3% frank pulmonary tuberculosis. He feels that preventoria for the observation of children who have been exposed to tuberculosis are needed as urgently as sanatoria for the treatment of adults with tuberculosis.

Kerr, R. B. (*N. E. J. M.*, Oct. 4, 1928, CXCIX-669) feels that the juvenile type of tuberculosis presents the golden opportunity for preventive work. He admits that its diagnosis is not easy and often cannot be positively confirmed. He emphasizes that one should not seek for pulmonary tuberculosis as evidenced by crepitations in the lungs but for lymphatic tuberculosis as shown by parasternal and paravertebral dullness.

(Practically no one will agree with Kerr as to the value of parasternal and paravertebral dullness.)

B. C. G. VACCINE

Calmette, A. C., Guerin, L., Negri, L., and Boquet, A., (*Rev. d.l. Tub.*, Feb. 1926, VII-5) after reviewing the entire subject of early attempts of prophylactic vaccinations against tuberculosis, give a detailed account of their work. They find that subjects free from all pre-ëxisting tuberculous infection can be artificially protected against natural or experimental infection by either inoculation or absorption of a culture of living, originally virulent bacilli, whose characteristics have been slowly and hereditarily modified through a long series of cultures on an alkaline medium, such as lipoids, so that it is harmless for mammals and birds. At first, under experimental conditions, they found that B. C. G. could effectively protect young cattle and apes against natural or artificial tuberculous infection for over a year. Following the favorable results obtained in animals, they have adopted this method apparently successfully in infants born of tuberculous mothers and found a mortality of only 2% as against 25% in the non-vaccinated. The duration of the immunity conferred appears to be long enough to guard young children up to the age of three years from family contamination, if the doses are not too massive. They practice revaccination at intervals of about a year, thus reinforcing the immunity. They found that this method of protection is harmless and results in no febrile reaction or physiological disturbance.

Calmette, A., Guerin, C., Negri, L., and Boguet, A., (*Presse Med.*, Feb. 24, 1926, No. 16-241) discussing the same subject, feel that this method is harmless and that its good results (a maximum of 8% against a minimum mortality rate of 25% in the non-vaccinated) warrant its general use, especially in newborn infants in a tuberculous environment. They feel, however, that vaccination will not permit the abandonment of hygienic measures necessary to prevent or minimize infection.

Calmette, A. (*Prog. Med.*, Jan. 21, 1928, No. 3-114) finds that anti-tuberculosis immunity produced by the B. C. G. lasts much longer than was expected at first. He finds that the immunity persists for at least five years. He also cites figures that prove the harmlessness of B. C. G. vaccination. He concludes that its efficacy can no longer be doubted.

Domingo, S. P., and Mirabelli, M., (*Rev. d.l. Tuberc.*, October 1927, VIII-668) after considerable experience with the B. C. G. vaccine, reported that no unfavorable reactions attributable to the vaccine occurred except in children who had evidently already been infected and in whom there were manifestations of intolerance. They strongly reaffirm Calmette's conclusions as to the effectiveness of the B. C. G. vaccine.

Beneden, J. (*Rev. Belge. d.l. Tuberc.*, Sept.-Oct. 1927, XVIII-235) found that the results obtained with the B. C. G. vaccine in Belgium, as in France, warrant its more extensive use.

Bergh, M. R. H. (*Wien. Klin. Wchnschr.*, Oct. 4, 1928, XL-1400) is of the opinion that the oral administration of B. C. G. vaccine confers a relative, but not absolute, immunity against tuberculosis. He, therefore, advocates revaccination at different intervals.

Weill-Halle, B., and Turpin, R. (*Arch. d.l. Med. d. Enf.*, Sept. 1928, XXXI-517) feel that the results they have obtained during the past six years warrant extending the method of vaccination to exposed infants and to infants not exposed, but whose parents desire the vaccination; also, to adults, especially those who care for the sick and are likely to be exposed to tuberculosis.

Nobel, E. (*Wien. Klin. Wchnschr.*, Jan. 19, 1928, XLI-798) found that vaccinated animals with the B. C. G. organisms were not immune to a subsequent virulent infection, except that most of them lived considerably longer than the controls.

Nobel, E., (*Wien. Klin. Wchnschr.*, Jan. 19, 1928, XLI-441) in co-öperation with Chiari and Sole, has shown that some animals inoculated with the B. C. G. vaccine develop progressive tuberculous lesions, also that tubercle bacilli can

be isolated from the lesions which are virulent. He repeats his emphatic warning against the general use of the vaccine.

Calmette, A., (*Wien. Klin. Wchnschr.*, Jan. 5, 1928, XLI-14) in answer to Dr. Nobel of Vienna, reiterates his findings that B. C. G. vaccine, even in large doses, never causes any more serious disease than swelling of lymph nodes and localized cold abscesses. He also warns that immunization by one subcutaneous injection is not deemed advisable in communities in which tuberculosis is endemic.

Petroff, S. A., Brench, A., and Stienken, W., Jr. (*Am. Rev. Tub.*, Jan. 1929, XIX-9) find that the B. C. G. micro-örganism in undissociated form is not very virulent for guinea pigs and rabbits, and that progressive tuberculosis occurs rarely, but it does occur. They feel, however, that the human body is not to be compared to glycerol bile-potato as a medium and that it would be hard to predict what may happen in the course of time if B. C. G. is implanted in the human body. Hence, they believe that the use of B. C. G. in prophylactic immunization may be a dangerous procedure at present. Nevertheless, they feel that further experiments should be carried on.

Calmette, A., (*Wien. Klin. Wchnschr.*, May 24, 1928, XLI-725) in answer to Petroff's experiments, which alleged a reversion of B. C. G. strain to virulence, finds it to be very unlikely, and claims that the experiments have been repeated in the Pasteur Institute with negative results. He reiterates that the effectiveness and harmlessness of the B. C. G. vaccination have been proved in 150,000 infants.

Schroetter, H., (*Wien. Klin. Wchnschr.*, Feb. 3, 1927, XL-151) after an extensive discussion of the literature, makes the following statements: The B. C. G. bacilli possesses constant properties both in vivo and in vitro. Even after the administration of large amounts of these bacilli orally, subcutaneously, or intracardially, no progressive disease develops. Whatever lesions may be found undergo complete resorption. The bacilli, which may be excreted after immunization, are harmless. Observations over several years have shown that the mortality of infants living in contact with open cases of tuberculosis has been reduced to one-twentieth.

Svarez, (*Wien. Klin. Wchnschr.*, March 24, 1927, XL-381) after injecting forty guinea pigs intraperitoneally with five to ten mgm. of the vaccine, found that the B. C. G. bacillus is nonvirulent and not tuberculogenic, thus agreeing fully in his findings with that of Calmette.

Calmette, A. (*Prog. Med.*, Jan. 14, 1928, No. 2-67) feels that it is legitimate to vaccinate with B. C. G. any individual at any age who is apparently well, and fails to react to tuberculin.

Selter, H., and Blumenberg, W. (*Klin. Wchnschr.*, June 11, 1927, VI-1134) on the other hand, working with the B. C. G. vaccine, produced a slight immunity but not sufficient to prevent reinfection. They found that Calmette's opinion, that B. C. G. strain induces immunity without reproduction of tuberculous alterations, cannot be accepted as correct. They found that the immunity lasts only as long as the initial lesion exists, probably too short a time for practical needs.

Kraus, R. (*Wien. Klin. Wchnschr.*, Dec. 1, 1927, XL-1508) feels that it is not known as yet how long the immunity caused by B. C. G. vaccine will last, and whether revaccination is desirable and safe. He sounds a warning against its use by general practitioners until further studies and experiments have been made.

Kraus, R. (*Wien. Klin. Wschnschr.*, March 29, 1928, XLI-441) likewise is of the opinion that, although the B. C. G. strain is apparently harmless, the general application to infant immunization at the present time would be premature.

Pirquet, C., (*Wien. Klin. Wchnschr.*, June 7, 1928, XLI-798) after reviewing the entire subject of vaccination with B. C. G., finds that immunization of children during the first years of life by active infection is dangerous and not promising and does not approve this method of vaccination.

Rosenfeld, S. (*Wien. Klin. Wchnschr.*, June 7, 1928, XLI-800) reviewing Calmette's statistics, finds that they contain grave errors, and that even without the correction of these errors an immunizing property of the vaccine has not been proved by statistics.

Chiari, H., Nobel, E., and Sole, A. (*Ztschr. f. Tuberk.*, July, 1928, L-24) warn against the immunization of infants with the B. C. G. organism and suggest that tests be limited to animal experiments for the time being. They do not consider the B. C. G. strain entirely avirulent but purely a micro-örganism of reduced virulence.

Baron J. (*Wien. Klin. Wchnschr.*, XXXII-1167) concludes that the apparent reduction in mortality in vaccinated children is chiefly due to the fact that they are under constant medical observation and that this reduction in mortality is not due to vaccination.

Greenwood, M., (*Brit. M. J.*, May 12, 1928, 3514-793) writing on a criticism of Calmette's statistics of B. C. G., comes to the conclusion that his reports cannot be regarded as a serious contribution to scientific literature.

PNEUMONOCONIOSIS

Pitchford, W. W. (*J. Industr. Hyg.*, April 1927, IX-109), writing on silicosis of the South African gold mines and the changes produced in it by legislative and administrative efforts, recognizes several clinical forms of silicosis: (1) simple silicosis, characterized by numerous small, widespread, symmetrical nodules of dense fibrous tissue in the lungs; (2) tuberculosis with silicosis where the nodules are less symmetrical in distribution and larger; (3) tuberculo-silicosis usually appearing as if the tuberculous infection were superadded to silicosis, but occasionally seeming to have first been pulmonary

tuberculosis (the latter called silico-tuberculosis). He found that by the end of seven years a little more than half the cases of simple silicosis have become tuberculous. The only early physical sign of simple silicosis found is a loss of elasticity of the chest wall and a high-pitched inspiratory murmur indicating lessened intake of air. The present compensation laws of South Africa classify silicosis as being of three stages:

(1) Ante-primary, when the earliest signs of the disease are or have been detectable, but before impairment of capacity to work has occurred.

(2) Primary, when signs are or have been present, and the working capacity of the individual is or has been impaired by the disease.

(3) He reports that a decided effort is being made to prevent the occurrence of silicosis and its complications by use of both mechanical and medical precautions, such as the prohibition of dry drilling or blasting during work hours, the provision of clean air, etc., initial and periodical examinations, special examinations, and attention to individuals requiring them, and a well-worked out scheme of organization of the Medical Bureau.

Hague, O. J., McBain, D. R., and McBain, R. W., (*Am. J. Roent. & Rad. Ther.*, October 1927, XVIII-315) studying silicosis as an industrial hazard in Ontario gold mining, found that the local incidence of silicosis is low in comparison with that of South Africa. They found tuberculosis the predominating serious factor, but not nearly to the same extent or violence as in South Africa.

McFarland, W. J., (*Industr. Hyg.*, Aug. 1927, IX-315) after reviewing the early symptoms as well as the complications of silicosis, reports the death rate from tuberculosis among the granite cutters as 1095.5 per 100,000 and that of the state at large only 96.4 per 100,000. He urges the practising physicians to pay more attention to the disease and the industry provide better dust-removing apparatus, etc.

Tathersall, J. (*Industr. Hyg.*, Nov. 1926, VIII-466) finds that the incidence of silicosis among coal miners varies a great deal, depending upon the type of coal, geological composition of the rock in the immediate vicinity, the wetness or dryness of the particular mine, and other factors. He urges that all men employed in this particular type of drilling should all be registered and have an initial and an annual physical examination. Those suspected of having tuberculosis should be excluded from the mines. He advocates the abolition of dry drilling.

Kettle, E. H., (*J. Industr. Hyg.*, Nov. 1926, VIII-491) studying the relationship of silicosis and tuberculosis experimentally in animals, found that the action of silica is a chemical one, brought about by hydrolysis of the silica in the tissues, the resulting soluble product being a protoplasmic poison. He found that silicotic lung tends to become tuberculous, not because it is fibrotic nor because there is interference with the drainage of lymph, but because of the presence of silica.

Hawes, J. B. 2nd, (*Am. Rev. Tub.*, Dec. 1928, Vol. XVIII-767) writing on tuberculosis as an industrial accident, clarifies the Massachusetts Workman's Compensation Law, especially in regard to chronic diseases like tuberculosis or pneumonoconiosis. He finds that the Massachusetts law includes under personal injury any injury or disease which arises out of and in the course of the employment which causes incapacity for work and thereby impairs the ability of the employee for earning wages, thus differing from the English Workman's Compensation Act which applies only to personal injury by accident. He finds that the interpretation of the court is that a personal injury must arise not only in the course of the employment but also it must arise from it.

HELIOTHERAPY

Laird, A. T. (*Am. Rev. Tub.*, July 1928, XVIII-80) makes a plea for a conservative but more infrequent use of heliotherapy in cases of pulmonary tuberculosis, although he admits that its results are not likely to be spectacular, and its omission is not necessarily detrimental to the patient's best interests.

Hernaman-Johnson, F. (*Brit. Med. J.*, March 26, 1927, 3455-562) feels that by cautious dosage (erythema should be avoided) and a careful selection of cases much good may be done by heliotherapy. The patients must not be very ill nor must they be of the well-known type which offers little resistance to the spread of the disease. The case that would struggle on for some years doing a certain amount of work is the one that will derive benefit. He further states that, if it is borne in mind that for the most part light acts as a stimulus to natural powers of defense, its limitations will be understood.

Bundersen, H. N., Lemon, H. B., Falk, I. S., and Coade, E. N., (*J. A. M. A.*, July 16, 1927, LXXXIX-187) studying the transmissibility of ultra-violet light through various mediums, found that vitaglass was far richer in transmitted radiation than other special manufactured transmitters of light, and compared well with quartz.

Wyman, E. T. (*N. E. J. Med.*, April 26, 1928, CXCVIII-510) feels that the average infant and young child in Boston gets insufficient sunlight during the winter months. He found sunlight when obtainable to be preferable to artificial light. However, he obtained good results from the use of lamps. He found also tuberculosis of mediastinal and mesenteric lymph nodes to respond well to ultra-violet treatment.

Strandberg (*J. A. M. A.*, May 19, 1928, XC-1595) reports that treatment by universal carbon arc irradiations is suitable in all cases of laryngeal tuberculosis. Reflected sunlight treatment of the larynx was unsuccessful because of the difficulty of irradiating the actual areas affected, and because the mirrors absorb too many

of the chemically active rays. Two hundred and three patients with laryngeal tuberculosis were treated by general carbon arc irradiations plus local treatment with the result that 55.6 of the cases were cured, hoarseness disappeared in 110 patients, and pain with aphagia disappeared in 25.

Howson, C. R. (*Cal. & Western Med.*, Jan. 1928, XXIX-25) concludes that heliotherapy in pulmonary tuberculosis is not harmless and that the dosage must be carefully graduated. He feels that it is not necessary to expose the chest directly to get ill effects. He found that exposure of the skin to the air alone is responsible for much of the benefit derived from the sun baths. He warns that we must continuously watch for unfavorable symptoms, such as increased temperature, weakness, dizziness, nausea, headache, increased pulse rate, anorexia or nervous irritability.

Boire, W. T., (*Boston Med. & Surg. J.*, Feb. 9, 1928, CXCVII-1509) making a comparative study of the effects of various forms of radiant energy and its therapeutic efficiency, found that the white flame arc, using 90 amperes, seems to best approximate the sun's luminosity. He found that at Flagstaff, Arizona, sunlight is nearly 50% more powerful than at sea level, Washington, D. C. Corex and fused quartz were found to be most efficient helioglass and vitaglass about 50% efficient, Cel-o-glass 20-30%, and other samples of glass almost nil.

Goodman, H. and Anderson, W. F. Jr., (*Boston Med. & Surg. J.*, Jan. 12, 1928, CXCVII-1316) found that the use of any special glass in city houses for the purpose of transmitting ultra-violet rays is of very doubtful value, as there is almost a total absence of "Vital" intraviolet lights in cities polluted with smoke and dust. In their study they found that fused quartz transmits 87% of the intraviolet light, Corex nearly 70%, Vioray about 56%, and vitaglass 42-49%.

MISCELLANEOUS

Geer, E. K., Callahan, and Gardiner, D. G., (*Am. Rev. Tub.*, April 1929, XIX-434) find that iodized oil can be used with safety in tuberculous fistulae as well as in tuberculous empyema cavities. They feel that it should be used for more exact diagnosis in unexplained hemoptysis and in unsuccessful thoracoplasty cases. They advise its use routinely preoperatively in thoracoplasty cases in order to guide the surgeon regarding preliminary phrenicotomy and as to the extensiveness of the contemplated rib resections. They advocate the supraglottal method in cases with chronic lesions.

Wynne, L. H. (*Lancet*, Nov. 5, 1927, CCXVIII-964) finds that apart from syphilis, tuberculosis and dust inhalation, there are many chronic diseases in which the lungs show signs of permanent damage in the form of fibrosis, more or less bronchial dilatation, and possibly thickened pleura due to influenzal infections. He found as a result of infection with influenza bacillus chronic bronchiolectasis, localized bronchitis, a combination of the above types with asthma, massive fibrosis, as well as bronchiectasis. He feels that the most important error in diagnosis is to mistake these cases for tuberculosis.

Knopf, S. A. (*J. Am. Med. Asso.*, Feb. 18, 1928, XC-532) going over the statistics of the tuberculous death rate in New York and Baltimore, finds the average annual decline of the male death rate has been much more pronounced than the decrease in the female death rate. Among factors responsible for this increased mortality among young women, he maintains, is the modern dress of women, the desire to have a slender figure resulting in undernutrition and unhygienic environment among office and factory employees.

(Most of us will not agree with Dr. Knopf's remarks concerning women's dress.)

McLaughlin, A. G. (*Lancet*, March 10, 1928, CCXIV-489) found that the main element of pleural pain, i.e., the sharp lancinating pain, worse on deep inspiration, is due to tension or stretching of the inflamed parietal pleura.

Goldberg, B., Sweany, H. C., and Brown, R. V., (*Am. Rev. Tub.*, Dec. 1928, XVIII-745), reporting on postmortem findings of gastro-intestinal pathological changes in patients who died of advanced pulmonary tuberculosis, find that the incipient lesions are found most frequently, first, in the lymphoid tissue above the ileo-caecal valve, second, in the caecum at the point where the food-current strikes the caecum, third, in the margin of the ileo-caecal valve, and very rarely at isolated points in the ileum or colon. They feel that concomitant diseases are not uncommon, some of which are related to the tuberculosis while others are not.

Opie, E. L., and Aronson, J. D., (*Arch. Path. & Lab. Med.*, July 1927, IV-1) studying the incidence of tubercle bacilli in latent lesions, found that latent fibro-caseous tuberculous lesions of the pulmonary apex in most instances contain living tubercle bacilli. Approximately one-fourth of fibrous scars of the apices contain living tubercle bacilli even though they do not exhibit gross or microscopic evidence of tuberculosis. They found only 9% of caseous, encapsulated, or calcified tuberculous nodules of lungs and lymph nodes produce tuberculosis when inoculated into guinea pigs, unless they are derived from lungs in which there is an apical lesion. They also found that lung tissue from the apex or base of the lungs, also tracheo-bronchial lymph nodes in which tuberculous lesions are not present, often cause tuberculosis when inoculated into guinea pigs. They found living tubercle bacilli in one or another part in slightly less than 50% of all persons. Living tubercle bacilli demonstrated by animal inoculation from caseous, encapsulated, or calcified lesions of lungs or tracheo-bronchial lymph nodes they found to be derived from the adherent lung or lymphoid tissue and not from the lesion itself.

VERMONT STATE MEDICAL SOCIETY

HOUSE OF DELEGATES, 1929

THE fixed session of the House of Delegates of the Vermont State Medical Society was called to order by the President, B. D. Adams, at five o'clock, Thursday afternoon, October 10, 1929, at the Woman's Club room in the City Hall at Bellows Falls, Vt.

Secretary J. E. O'Brien called the roll, and

PRESIDENT GEORGE R. ANDERSON

by vote of the House vacancies therein were filled, making the Roll of the House as follows:

Bennington:—Henry Goodall, L. H. Ross.
Chittenden:—B. D. Adams, G. M. Sabin, C. H. Beecher.
Franklin:—Edwin A. Hyatt.
Lamoille:—W. M. Johnstone.
Northeastern:—F. E. Farmer.
Rutland:—C. F. Ball, J. E. O'Brien, H. L. Frost, W. H. Grinell.
Washington:—C. H. Goyette, J. A. Wark, R. A. Russlow, F. C. Angell.
Windham:—F. L. Osgood, C. S. Leach, J. A. Stevenson.
Windsor:—A. M. Cram, B. A. Chapman.

By vote of the House the reports of the secretary, treasurer, and auditor were accepted as printed; also the reports of the Publication, Medico-Legal, and Necrology Committees, and the report of the New England Medical Council.

Under Unfinished Business it was voted to amend Article IV—(Meetings) of the Constitution to read:

There shall be an annual meeting of this Society held in October at such place as shall be designated by vote of the House of Delegates at the previous annual meeting.

Under New Business:

(a) It was voted that Burlington be the place of the next annual meeting.

(b) Dr. C. H. Beecher, by request, offered the following resolution:—

Resolved, That the House of Delegates endorse as an addition to the Compensation Law the words:—

"That the employee is entitled to be treated by the physician of his own selection"

which resolution was adopted.

Voted that the House of Delegates authorize the sum of $100 to be paid to Dr. J. N. Jenne, for his expenses as representative of the Vermont State Medical Society at the meeting of the A. M. A.

Dr. Beecher moved: That Article VII of the By-laws be amended, so that the last line shall read *shall be published annually;*

That Article VIII of the By-laws be amended so that the last line shall read *shall be printed annually;*

That Article X of the By-laws be amended by substituting the word *Records* for the word "Transactions";

That Article XV be amended by substituting the word *Records* for the word Transactions;

All of which proposals of Amendment were voted.

Dr. E. A. Hyatt offered the following resolution:

Resolved, That we approve an attempt to arrange with the Massachusetts Medical Society to own and operate the NEW ENGLAND JOURNAL OF MEDICINE, through the New England Medical Council, if agreeable to the Massachusetts Medical Society, when and if four other New England States join in the effort;

which resolution was adopted by the House of Delegates.

Dr. Burbank offered the following resolution:

That it seems advisable that a State Medical Society Councillor see the doctors in Barre whose names are posted on the insurance company cards, advising that it is not in the spirit of this Society; That they be asked to have these cards removed because no name can be used without one's consent and approval; and that it is unethical, according to the Code of Ethics of the American Medical Association to continue to allow this card to be posted;

which resolution was adopted.

Under Election of Officers, Dr. F. L. Osgood

nominated Dr. George R. Anderson, of Brattleboro, for President for the ensuing year. Nomination seconded by Dr. J. A. Stevenson. Dr. Anderson was then elected President by vote of the House of Delegates.

On motion of Dr. J. A. Stevenson, it was voted that the President of the House appoint a nominating committee of three to bring into the House of Delegates, at its adjourned Friday morning session, nominations for all the other offices, committees and delegates of the Society; also nominations for officers of the House of Delegates.

President Adams appointed as such nominating committee, Doctors J. A. Stevenson, C. H. Beecher, and W. M. Johnstone.

On motion of Dr. Osgood the meeting adjourned until Friday morning, at 8:30, same place.

Friday Morning Session

The adjourned meeting of Thursday, October 10, 1929, of the House of Delegates reconvened in the Woman's Club room of the City Hall at Bellows Falls, Vt., Friday morning, October 11, 1929, at 8:30, with President B. D. Adams in the Chair.

Meeting called to order by President Adams.

President Adams called for the report of the nominating committee.

Dr. J. A. Stevenson, reporting for the nominating committee, submitted the following nominations for the officers, committees and delegates of the Society:

President: George R. Anderson, Brattleboro.
Vice President: W. M. Johnstone, Morrisville.
Secretary: William G. Ricker, St. Johnsbury.
Treasurer: David Marvin, Essex Junction.
Auditor: George M. Sabin, Burlington.
Anniversary Chairman: John Woodruff, Barre.
Executive Committee: W. G. Ricker, C. H. Beecher, C. F. Ball.
Publicity Committee: W. G. Ricker, C. F. Dalton, J. A. Wark.
Medical Education: Angus Black, S. W. Hammond, A. B. Bisbee.
Necrology Committee: L. A. Russlow, B. D. Adams, H. S. Pierce.
Medico-Legal: J. N. Jenne, E. A. Hyatt, A. L. Miner.
Health and Public Instruction: P. E. McSweeney, B. D. Adams, A. L. Patch, C. F. Dalton, P. K. French.
Legislative: J. H. Blodgett, L. H. Ross, A. L. Patch.

Delegates:
Maine: C. H. Beecher.
New Hampshire: John H. Blodgett.
Massachusetts: C. F. Dalton.
Rhode Island: E. A. Tobin.
Connecticut: A. L. Patch.
New York: J. N. Jenne.
American Medical Association: W. G. Ricker.
American Medical Association: C. H. Beecher (Alternate).
New England Medical Council:
George H. Anderson
William G. Ricker
E. A. Hyatt
C. F. Dalton
J. A. Stevenson.

On motion of Dr. Beecher the Secretary cast one collective ballot for the officers, committees and delegates nominated for the Society, and President Adams declared them elected to the respective offices, committees, and delegations.

Dr. Stevenson, for the nominating committee, submitted the following nominations for the officers of the House of Delegates:

President: L. A. Russlow, Randolph.
Vice President (1): J. E. O'Brien, Poultney.
Vice President (2): Charles A. Ravey, Burlington.
Secretary: J. A. Wark, Barre.

On motion of Dr. Beecher the Secretary cast one collective ballot for the officers nominated, and President Adams declared them elected.

On motion of Dr. Beecher, seconded, the report of the nominating committee was accepted.

On motion of Dr. Stevenson, seconded and carried, President Adams declared the meeting of the House of Delegates adjourned.

COMMUNICABLE DISEASE PROBLEM*

BY CHARLES F. DALTON, M.D.†

ONCE in a while there comes to a man an occasion of which he may justly be proud. Such an occasion is that of being elected to preside over the medical society of his state, and be thereafter recognized as a president of the organization.

At the very beginning of this address, I desire to express my great appreciation of the honor which has been mine during the last year and to thank the physicians of Vermont for their hearty co-öperation in the efforts which have been put forth for the growth of the society and the unification of the profession.

This has been a good year in many ways. Perhaps, for the first time, the president and the secretary have visited every county society, and an acquaintance thus established which could not have been effected in any other way. It was the opinion of all that such visitations and the accompanying special lectures ought to be made an annual feature.

*President's Address read before the Annual Meeting of the Vermont State Medical Society, Bellows Falls, October 10, 1929.

†For record and address of author see "This Week's Issue," page 843.

Death has removed several of our members whose places can not be filled. These vacancies must serve to remind us of the medical service problem which is gradually growing in our state and which some time must receive recognition by the State Medical Society. The reports of the Secretary and the chairmen of Committees show in detail the present satisfactory condition of the Society, so that it is neither appropriate nor necessary for me to discuss it at this time. It is my desire to take up with you certain problems connected with communicable diseases, which loom large to me, probably because for several years my work has had to do with this phase of practice.

Every practicing physician, whether doing general or special work, at some time comes in contact with communicable diseases, and therefore is concerned in the problems relating to their diagnosis, treatment and control. Every physician is familiar with the oft-repeated inquiry, "How is this disease caught?" and every physician knows how he attempts to explain the method of transmission in terms understandable to the laymen, or else frankly confesses that he does not know. Every physician recognizes the difficulty encountered in endeavoring to make a diagnosis on an early case, and, according to his disposition, registers chagrin or amusement when he realizes the mistakes he has made. Fortunately in the average intelligent family, these problems may be explained and understood, but even in the best families there lurks a feeling that if the doctor is not able to immediately label the disease and answer the inquiries concerning it, there is something lacking in the medical profession which ought to be remedied. And to this idea we must, on reflection, inevitably subscribe.

There is the problem of scarlet fever. Five years ago when the Dicks announced their findings on the hemolytic streptococcus and there followed the production of specific serum, together with methods for active and passive immunization, the Dick test for susceptibility and the blanching test for diagnosis, it seemed as if our troubles with this disease were over. We, in Vermont, induced the Legislature to include the distribution of scarlet fever serum along with diphtheria antitoxin for the use of the doctors, and the law is now on the books. But consider the situation as it has developed. The active immunization, even after the administration of several and increased doses of the toxin turns out to be of only temporary value, lasting at the longest probably not more than a year, so that the routine preventive treatment of school children is entirely impractical. The serum which we hoped would prove the partner of diphtheria antitoxin, causes so many serious reactions that doctors hesitate to give it except in the most severe cases. The blanching test requires a technique which few doctors possess.

In the laboratory, routine examination for the streptococcus scarlatinae is not a practical procedure, so that we have no way of determining when a throat is clear of organisms or whether a convalescent case remains as a temporary or possibly a chronic carrier. Meanwhile we quarantine the case for four weeks and if clinical features of scarlet fever have disappeared, we release the patient and hope that secondary cases will not occur. Not infrequently a case of German measles goes into quarantine under the diagnosis of scarlet fever and the public can not understand why the doctor fails to discover the difference.

Measles presents a problem which is still unsolved in spite of the many thousands of cases which occur, and that problem is, in brief, how to control the disease. There seems to be some hope that the causative factor may in time be recognized, and, when that time comes, there may be some means of immunization. At present, what do we do? We quarantine the case, usually after the eruption appears, and we well know that the most infectious period is during the catarrhal stage. We give immune serum in institutions. We try to instruct parents and school teachers to exclude from school all children who exhibit symptoms of acute coryza. We attempt to isolate contacts during the later days of the incubation period in the hope that they will come down in private rather than in public. Yet every health officer knows that, try as he will, an epidemic of measles will go on in spite of all his efforts. Fortunately the disease, if properly watched, seldom proves fatal in children over five years of age; consequently strenuous efforts are now being made to protect the little ones, and there is a tendency to relinquish precautions regarding the older children. There is serious doubt as to whether this method gives the public the protection to which it is entitled, but, on the other hand, what else can be done? The answer may be forthcoming, but it has not yet been satisfactorily given.

Then there is the problem of poliomyelitis, best known to the public as infantile paralysis. In 1913 Noguchi described a small globoid organism which for a time was accepted as the active cause of the disease. At other times various strains of streptococci have been recognized as the apparent cause, but after all these years we have to content ourselves with the statement that the infecting agent is a "filtrable virus." The method of transmission of poliomyelitis has been variously believed to be nose and throat secretions, fecal and urinary discharges, infected milk, infected fleas from rats, other biting insects, dusts of various kinds, and contact with paralyzed domestic animals. In fact almost every known method has at some time been suspected, and to some extent investigated. Some of the hypotheses appear to be tenable while others require scant scrutiny before being dismissed.

The diagnosis of poliomyelitis in the preparalytic stage is shrouded in mystery and fraught with many pitfalls. The average practitioner simply admits that he only suspects the disease, and if it is an early case in a series, he may not even suspect it until paralysis forces the diagnosis upon him. As to treatment, there is little to do, unless spinal puncture is resorted to, and even then the administration of immune serum is of questionable value.

The health department meanwhile wrestles with the subject of quarantine and control. We say that the public must be protected, must be given the benefit of the doubt, and we enforce a quarantine of three weeks. None but those who have actually dealt with the problem can realize how often arise the questions; "Is such a quarantine necessary?" "What shall we do with the contacts?" "Should the quarantine include the whole family, or does it simply require the isolation of the patient?" We have yet to be shown that any of these procedures have any deterrent effect upon the incidence of the disease, but, in the light of our present knowledge, the methods we now use appear to be necessary for the protection of the public and must be accepted in that light.

The Vermont State Board of Health has been continuously studying this disease since 1914. We have seen other institutions and organizations launch out into intensive research, only to give up their efforts after repeated failures. We have recently seen a renewed interest in this vexing problem, and today a number of agencies are concentrating on the work of solving the mystery. Let us hope that science will soon break through the darkness in which we are groping and give us the light of definite knowledge.

To many physicians, it may appear that typhoid fever has been conquered; that it has reached an irreducible minimum. If one consults vital statistics and compares the low death rates of recent years with those of a generation ago, such a conclusion might seem to be justified. Yet there is still a problem connected with typhoid fever, and that problem is the discovery and control of the chronic carrier. How is the man on the street to know whether he is being infected with typhoid by the man who sells milk to his family, by the waitress who serves him in the restaurant or hotel, by the cook who prepares the food in his kitchen, or by the friend who invites him to a meal at his home? How is the Board of Health to weed out these persons who are a menace to life and health wholly through their careless habits? And when the carrier is discovered, what is to be done with him? We believe that universal immunization will control the incidence of the disease and history shows that it will. We believe that proper pasteurization of all milk will prevent milk-borne epidemics and it will; but pasteurization is not a practical procedure in rural sections. Water-borne typhoid is so rare in this state that it is practically negligible, but the carrier is with us. Evidently some method of isolation and maintenance must be worked out before the carrier problem is solved. Until this is done we must depend upon continued instruction of the public in the simple lessons of common, decent cleanliness.

The venereal diseases present a problem which is mainly one of administration. The public, having been for many years impressed with the idea of secrecy and concealment in these cases, cannot readily be aroused to bring them into publicity. State laws provide for reporting venereal diseases, but no state presumes to boast of 100% reports or even anything near that. Human nature in this case overrules law. But suppose we have 100% reports, or 75%, or 50%, what should we have gained? From an educational standpoint, we should have impressive figures to give out to the public as a warning against a menace to oneself and family. But what shall we do with the cases? They cannot be regularly quarantined, like scarlet fever. Most of them must go on with their daily work, but they must receive treatment. The problem, then, is treatment. Treatment for the negligent case who believes himself cured when acute symptoms disappear; for the floater, who goes from one doctor to another, and in consequence misses the benefit of consecutive supervision; for the dependent case which no one wants to treat; and for the case which does not get to the doctor, but takes quack medicine on the advice of friends or as a result of fraudulent advertising; these are the problems. With only one hospital in the state willing to receive the venereal patient as a ward case what can town officials or even courts do? What can the Board of Health do?

Fortunately, in this state, there are many physicians who are willing to treat these cases without pay and this to a great extent solves the problem. But not wholly. Before venereal diseases can be controlled and adequately treated, there must be radical changes not only in the law, but in public opinion and these changes may not come during the present generation. This problem presents a somewhat different aspect from those previously cited, in that every physician may contribute to its solution through the medium of the education of his own patients. Instruction as to the amount and kind of treatment needed and the dangers of neglect, if uniformly given by members of the State Medical Society, could not fail to make some impression on this serious problem.

These problems are only a few of those which present themselves in the handling of communicable disease. Many others occur and new problems are constantly arising. Some are due to the difficulty of discarding discredited theories, while others arise from lack of adequate knowledge. The first difficulty will be remedied when physicians avail themselves of the information which research has demonstrated to be at least

practical and probably correct. Lack of knowledge can only be corrected by further study and to this end the medical profession is working as never before.

The best minds have been set to the solution of the difficulties presented by these problems and progress has been almost unbelievably great. The story of diphtheria shows what has been done in one disease, and what probably will be done in others. Your attention is simply directed to the wonderful field still open to those who have the mind to work, the heart to try, and the patience to continue the research which must be carried out before we have conquered those diseases which we are pleased to call "preventable."

Men and money are needed if the State Board of Health is to deal adequately with these problems, and some of them are not even within the range of a health department such as ours. One suggestion has been made that a committee be appointed, or some standing committee designated, to which the State Board of Health could go as a medium between it and the Legislature. The duties of such a committee would be to inform itself as to the need of medical things in the state, and to take such matters directly to the Legislature when more law or more money is required. The health department is the servant of the profession as well as of the public and the more the physicians understand the need of work and the frequent difficulties encountered, the greater will be the service rendered to all.

LEAD POISONING

(Continued from Page 819)

sirable. But, as stated above, there may be danger in too rapid elimination, therefore the treatment should be supervised by a competent physician.

In lead poisoning, as in other conditions, prevention greatly outweighs cure in importance. The responsibility for the prevention of industrial poisoning rests largely on the manufacturer. The worker, however, is also responsible. There are many instances in which the safety measures and devices that are provided are ignored or misused by the employee. The manufacturer should provide adequate safety appliances and educate the employee in their use as well as the results of their abuse or disuse, but it devolves upon every worker to help in making the shop a safe work-place, and to minimize the chances of the other fellow, as well as himself, getting dangerous doses of the poisonous substance. Particular care must be used to avoid dustiness, since the most dangerous cases of lead poisoning are those in which the lead has been breathed in. Personal hygiene and habits are factors in health in any occupation, but they are especially so where the worker is exposed to industrial poisons and other unfavorable conditions in industry.

MEDICAL CENTER PROJECT OUTLINED

The formation of a New England Medical Center, which will care for the sick and needy, young and old, and train family doctors for the small communities of New England, was announced October 16 at a luncheon in the Chamber of Commerce, attended by trustees of the Boston Dispensary, Tufts College and the Boston Floating Hospital, the institutions behind the project.

Plans have been drawn for the set of buildings which will be erected on the present site of the Boston Dispensary on Bennett Street.

When completed, the Medical Center will consist of a city dispensary, a modern free hospital for babies and small children, classrooms and clinical teaching facilities for the training of doctors, and scientific laboratory X-ray and operating equipment for the use of all three. The combination of medical treatment, hospital work, research and teaching will be thus realized.

The training of family doctors will be an especial feature it is claimed. By the erection of a modern children's hospital building, the Boston Floating Hospital will resume its work of caring for the sick poor babies of Boston, curtailed in 1927 when the hospital ship was burned.

For the project the present building of the dispensary will be remodeled.

To carry out these plans, it will be necessary for $970,000 to be raised for the buildings and $530,000 for endowment to provide additional income necessary to the three institutions. The total amoun necessary is $1,500,000.

A joint administrative board to carry out the plans for the new medical center will consist of Earle P. Charlton of Fall River, John A. Cousens, president of Tufts College; Charles R. Gow, postmaster of Boston; Ralph Lowell of Lee, Higginson & Co.; John R. Macomber of Harris-Forbes & Co.; Robert W. Maynard, president of R. H. Stearns Company; Francis S. Moulton of Pillsbury, Dana & Young; James Nowell of Lee, Higginson & Co; Edward W. Pope, chairman of the Boston Floating Hospital; Arthur G. Rotch, president of the Boston Dispensary; Henry B. Sawyer of Stone & Webster, and R. Minturn Sedgwick of Scudder, Stevens & Clark.

Among the services which the Floating Hospital will render will be extensive research in children's diseases, the training of about 135 nurses yearly, and a program for educating mothers in the care of their babies.

In addition to training 250 third-year and four-year medical students yearly, the Medical Center will give informal graduate instruction to older practicing physicians.

NEW OFFICERS OF DELTA OMEGA

At the annual meeting of Delta Omega, the honorary public health society, held in Minneapolis on October 2, 1929, during the convention of the American Public Health Association, C. C. Young, Ph.D., Dr. P. H., Director of the Bureau of Laboratories of the Michigan State Department of Health, was elected national president. John A. Ferrell, M.D., Dr. P.H., of the Rockefeller Foundation, New York, was elected national vice-president, and James A. Tobey, Dr. P.H., of New York, was re-elected national secretary-treasurer.

Case Records
of the
Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY R. C. CABOT, M.D.

F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 15431

ALBUMINURIA AND UNEXPLAINED SEPSIS

MEDICAL DEPARTMENT

An American schoolboy of nineteen entered February 27 complaining of swelling of the legs, ankles and feet.

All his life he had been ten or fifteen pounds underweight and less strong than other boys. He had always had a fondness for warm milk fresh from the cow, and had been in the habit of drinking a great deal of it. For five years he had had night sweats; none for the past month. For three years he had urinated once or twice at night. For the past year moderate exercise had made his abdomen feel tired and weak. For four months he had felt slightly feverish in the afternoons. Two months before admission physical examination at school showed that his temperature was 100.2° and his weight 118 pounds. The school authorities sent him for a thorough medical examination, including two X-rays. The findings were entirely negative. Seven weeks before admission he had an attack of grippe keeping him in bed for a week with a maximum temperature of 103° and a bad cough. A week later he had nosebleed three or four times a day for four days, severe enough to require medical treatment. His physician treated him for calcium deficiency. Since that time he had had a few mild nosebleeds. Five weeks before admission, after walking a little more than usual, his feet, ankles and shins were swollen at night. His physician put him to bed and gave digital pills and a diet. He followed the régime. Albumin and sugar were found in his urine. For five days he had had five or six watery movements a day with tenesmus. For the past month or two he had slept soundly ten or twelve hours at night.

One grandmother died of heart trouble; the other possibly had it. His mother had had three miscarriages, one following the birth of each of the first three of four living children. One uncle was in a tuberculosis sanitarium. The patient had had no association for any length of time with any tuberculous patient.

He had some tonsillitis before he was four years old. At seven years he had his tonsils and adenoids removed. His nose had always bled on slight trauma. He had had chicken pox, measles, pertussis, and at ten years influenza.

Clinical examination showed a poorly developed and nourished boy with pale skin and drawn features. Mucous membranes pale. No axillary or pubic hair. Teeth slightly carious. Gums of upper front teeth red and spongy. Soft palate injected. Thyroid isthmus palpable. Glands hard in the right groin. A few small glands in the neck and axillae. Chest flat. Left chest fuller than right. Expansion good and equal. Lower chest bulging. At the right apex in front slight dullness, increased tactile fremitus and prolonged respiration. Apex impulse of the heart seen and felt in the 5th space 7 cm. from midsternum, coinciding with the left border of dullness one cm. outside the midclavicular line. Blood pressure at admission: standing 95/65, lying down 125/75. Pulses and arteries normal. Abdomen distended, high pitched tympany, a doughy feeling, questionable shifting dullness and fluid wave. Much voluntary spasm. Slight tenderness deep in the left upper quadrant. Liver dullness two fingerbreadths below the costal margin. Edge not felt. Genitals very small for his age. Marked edema of the feet, ankles and legs. Pupils and reflexes normal.

Urine before operation: amount normal when recorded, specific gravity 1.036 to 1.028, a trace to a large trace of albumin at all of six examinations, no sugar, acetone or diacetic acid, 1 to 25 leukocytes at two sediment examinations. Renal function 20 to 50 per cent, two tests. Blood: 13,250 to 16,900 leukocytes, 61 to 64 polymorphonuclears, 5 myeloblasts at the entrance examination, hemoglobin 55 to 60 per cent, reds 4,350,000 to 3,700,000. Smear normal at admission. March 6 a smear showed the platelets and reds normal except for slight achromia. Hinton negative. Non-protein nitrogen 33 mgm. Blood sugar 83. Total protein 5.26 per cent. Stools: guaiac strongly positive at two of four examinations. Basal metabolic rate + 18.

X-ray examination showed the diaphragm rather high on both sides, sharply outlined, moving normally with respiration. The hilus shadows were moderately increased. There was no definite pathology in the chest. Examination with a barium enema showed the colon slightly large. There was considerable gas and feces in the colon, making examination unsatisfactory. No organic pathology could be made out.

Before operation temperature 97.7° to 101.4°, pulse 80 to 120, respirations normal except for one rise to 32 on March 4.

March 4 the edema of the legs had disappeared, leaving the abdomen as distended as ever. An abdominal tap was dry. The abdomen remained tympanitic in spite of catharsis. The shifting dullness persisted, although the fluid wave disappeared. Alpine lamp treatment was started.

March 20 a lymph node was removed from the

mesentery. The patient made a good recovery from anesthesia, but the next day was nauseated and uncomfortable, with some abdominal distention, diarrhea, and slight elevation of temperature and pulse. He continued to run a slight temperature all the time, with evening rise. March 27 he had a severe nosebleed. April 1, after he had been up in a chair, his ankles swelled as they did before he came into the hospital. X-ray treatment was given. April 4 the leukocyte count was 25,000, 73 per cent polymorphonuclears, 4 per cent unclassified, several abnormal white cells, occasional Türck cells. The platelets seemed much increased and occurred in enormous masses. Review of the previous smears now showed atypical mononuclear cells. The edema was controlled fairly well by postural treatment. April 11 there was considerable pleural effusion on the right.

April 12 the patient was discharged unimproved. April 16 he died at home.

Discussion

BY CHESTER M. JONES, M.D.

NOTES ON THE HISTORY

This boy's less than normal weight and strength may be due to living habits, may be due to the economic situation of the family. We do not know whether they have been able to supply him with enough food to enable him to develop normally.

I do not know just why the note that he was fond of warm fresh milk was put in, but it is worth a little comment. If he has had this fondness since infancy it is entirely possible that he may have been exposed to bovine tuberculosis. We do not know anything about the herd from which the milk was obtained. Presumably it came from different cows at different times. This habit may have a certain etiologic importance.

A history of night sweats is as valuable as the intelligence of the patient who gives it. I take it that the house officer or student who took this history believed the patient had real night sweats, drenching sweats, sufficient to soak the night clothes or bed clothes with perspiration. Night sweats continuous over a period of five years are however very unusual. We get a continuous sequence of night sweats in many acute infections of a serious nature, but they are of shorter duration than this. Five years is a long time. It is almost inconceivable that he could have had a disease active enough to cause night sweats continuously for five years and have him still living. Certainly one of the things most to be considered as a common cause of night sweats is tuberculosis of some sort. But it is surprising if the patient has had night sweats over a period of five years continuously. We have to surmise that it means nearly all the time. That they should disappear during the past month is rather surprising. In ordinary sweating from undernourishment or weakness of some kind that is not uncommon; but with real night sweats it is extremely uncommon.

In a young person I should say that frequent urination at night is rather extraordinary. It may be because he was awakened by the night sweats. Maybe when he got up to change his clothes he also got up and voided. It may be he actually has disease in the urinary tract, possibly nephritis with nocturia. No mention is made of painful micturition, therefore cystitis or urethritis would seem out of the question without knowing more of the past history. For the moment we should have to say that it may be a nervous phenomenon or may be associated with renal disease.

That "moderate exercise made his abdomen feel weak," that the weakness should be localized to the abdomen, is rather queer. Perhaps he is conscious of his abdomen and that is where he localized his pain. It may mean abdominal disease.

If he had real night sweats even for the past year he might have been running an afternoon temperature. Some people are not conscious of fever until the temperature is elevated to 100° or 101°. Other people are very conscious of it even when it is in the low ninety-nines. Night sweats, afternoon fever certainly make one think a little more of tuberculosis. They are consistent with the findings in tuberculosis of one type or another.

Presumably one of the X-rays taken was a chest plate. The other was possibly of the teeth and sinuses. They certainly must have thought of tuberculosis in a young person, then thought of some other focus of infection, difficult to find localized, either in the sinuses or the teeth. The findings were entirely negative. We cannot argue much about that as we do not know what they did. If there was a negative chest plate, that is certainly against pulmonary tuberculosis. But all one can do is guess a little.

The finding of fever, possibly morning fever, makes one wonder whether he did not have a higher temperature in the afternoon. It would fit perfectly with a sensation of afternoon fever and night sweats and be confirmatory evidence of tuberculosis.

I presume what is meant by "grippe" is any ordinary upper respiratory infection associated with a good deal of malaise. In this particular instance he was in bed for a week and may have been actually running an elevated temperature for the entire week. The symptoms of superimposed infection may have been part of a general process. But I should imagine this was merely a temporary affair.

I cannot explain the nosebleeds at all unless he has coryza. Epistaxis may occur when there are dry mucous membranes and injected veins if you blow hard enough.

I do not know what his physician found, therefore it is not fair to criticize. I should say there is nothing here to go with calcium deficiency.

The cause of continued nosebleeds may be still the same, the acute upper respiratory infection. They may be due to a local vascular injection having nothing to do with the remainder of the symptoms of which he complains.

Swelling of the feet is a new symptom, obviously of a good deal of importance, because edema in young people is practically always associated with definite organic disease. The most common cause of course is nephritis. Cardiac edema could be of exactly the same type. This came on after walking. It could be cardiac edema, but we have no other evidence at present of heart failure sufficient to cause cardiac edema. He has been a little short of breath, not able to do quite so much as other boys. It does not however sound quite like heart disease to me. Endocarditis continued over a long period may be associated with night sweats. Maybe he has endocarditis, although I am inclined to think the symptoms are not fundamentally cardiac in nature.

The doctor evidently thought he was cardiac. He had the privilege of examining him, which we have not. Rapid rate is not sufficient evidence for heart disease. Edema in a young person is not sufficient evidence for heart disease. The history of tonsillitis gives a possible background for heart disease.

We do not know the nature of the diet prescribed.

We do not know how much albumin was found in the urine. It is the first evidence definitely in favor of renal involvement rather than cardiac failure. In any case in which we have congestive heart failure we frequently have albumin in the urine. The amount of albumin depends on the degree of heart failure. The evidence of albumin is more in favor of renal disease than of cardiac. It may be that the sugar helps to explain the nocturia. No frequency is noted, however, by day, an element which will not check up with true diabetes. I do not see any reason for considering another diagnosis. We need more evidence before we add diabetes to the list of possibilities. Nervousness alone will give glycosuria. His feet were swelling and he became more or less frightened. It is not uncommon to find sugar in the urine in small amounts in men before examinations.

"For the past month or two he had slept soundly." That includes I should say the last five days.

Usually diarrhea with tenesmus in which there are no movements at night makes one wonder whether there is organic disease as the basis of the diarrhea. But the history this boy presents of various symptoms is very suggestive of organic disease; so probably we have to assume that the diarrhea is on the same basis.

Stopping with the history, it seems we have evidence of chronic infection associated with fever, probably true night sweats, loss of weight or inability to gain weight, acute cough, without striking heart symptoms, now with edema, and now for a few days with diarrhea. Tuberculous involvement of glands in the abdomen, primarily the mesenteric glands, could give such a picture. It is a little difficult to explain the edema on such a basis. We are dealing here with a disease going on for some years with apparently no break in the history, if the history is correct. One can have edema if there is enough disease in the abdomen, enough involvement of glands. One also can have albumin. Very rarely we see a condition of amyloid disease involving the kidney and liver. It may be the cause of the condition here. Bright's disease would account for night albuminuria. Mesenteric glands could account for diarrhea. The combination of tuberculous glands in the abdomen plus possible amyloid disease is the most logical explanation of the story.

I should say the sequence of events in connection with the mother's miscarriages is of no importance. The miscarriages alternated with births of normal children; therefore we have no evidence to make us believe his mother had syphilis, tuberculosis or other organic disease.

The history of his uncle's tuberculosis may or may not be of importance, because the patient says he did not associate with him. But it is possible that this boy was exposed to tuberculosis in infancy or childhood, did have tuberculosis, but had enough resistance to handle the situation at that time; later, because of undernourishment or something else it lighted up again. I do not believe however that the probability of exposure to tuberculosis is very important.

It may be he had enlarged veins along the nasal septum which bled very easily. I do not believe his tendency to bleeding at the nose has anything to do with the main diagnosis.

This is not an unusual past history. It is rather less than the average ward patient has as a background. I should think the past history is pretty much out of the picture.

NOTES ON THE PHYSICAL EXAMINATION

He was possibly anemic, although his pallor may have been due to the fact that he has not been out-of-doors. It may have been due to circulatory disease.

I do not know the significance of lack of axillary or pubic hair in this case. It probably goes along with the story of undernutrition. I do not think we shall find endocrine disease as such. If the diagnosis of tuberculosis is correct, one wonders if there is involvement of the adrenals preventing proper development. That is probably a wild guess.

"Red and spongy gums" is not surprising in view of the story of undernutrition. It may be

due to local sepsis. I do not think it means anything more than that.

The thyroid isthmus is palpable probably because he is thin.

We have a definite clinical finding of what appears to be an abnormal gland in the right groin. If it was very hard we wonder if it was calcified; calcified glands nearly always mean tuberculosis.

The fullness of the left chest may be due to some effusion. We do not know where the chest bulges. If it bulges in front we have to consider the possibility of heart disease with a very large heart. The rest of the history does not support that. The signs are not those of fluid. The bulging may be simply due to poor posture; it may be due to scoliosis.

The signs at the right apex may be physiological or they may be slightly increased over normal. In a very thin person it is more easily demonstrated than in a well-developed person. I should say, however, in view of the history, that we have no evidence of pulmonary tuberculosis except the findings at the right apex.

A midclavicular line of 6 means a very long and narrow chest for a boy of nineteen and I wonder whether that means enlargement of the heart or not. The apex impulse should correspond with the midclavicular line in an adult. I think, however, this boy is probably of the ptotic type. We should expect to find his apex inside the midclavicular line. This is simply a way of saying that the heart was in a more vertical axis than usual. One might say there may have been slight enlargement of the heart.

Very poor vasomotor tone; that is about all you can deduce from a change in blood pressure from the reclining to the standing position. Even in a good athlete there may be a slight drop. We know this boy's muscular tone is very poor, particularly of the leg muscles. He undoubtedly would have fainted if he had had to stand very long. Nothing more is said about his heart, therefore there is no evidence of heart disease, no reason to suppose the edema we found before or the albuminuria is at all related to disease of the heart.

The report on the abdomen sounds as if there were probably fluid in the belly with probably some distention in the bowel. "Questionable shifting dullness and fluid wave."—I suppose the adjective applies to both. Whoever made the examination did not feel sure that he had absolutely demonstrated fluid in the abdomen. But in a very thin patient one ought to be able to say that there is or is not fluid in the abdomen. These findings are very suggestive of fluid. I should think he probably had it. In adults the most common cause of ascites is cirrhosis. Other common causes are diseases that affect the peritoneum or the mesenteric glands. Tuberculosis of course is one of these. The diagnosis of tuberculosis may be entirely consistent with the presence of free fluid in the abdominal cavity. Occasionally in severe nephritis we get ascites, but it is usually a terminal event. He has not heart failure or signs of adherent pericardium so far as I can see. The glandular involvement seems to be due to nephritis with peritonitis.

The liver may or may not be enlarged. It may simply be low. We do not know anything about the upper border of dullness, so it is not fair to say the liver is large.

"Genitals very small." He is undeveloped. I should say that this is not an important finding.

The physical examination, then, shows that this boy has adenopathy, probably ascites, peripheral edema, no evidence of heart failure, no evidence of pulmonary tuberculosis.

There is one other thing to be considered here as capable of giving this clinical picture, that is so-called nephrosis, tubular nephritis. The clinical picture is not inconsistent, but I do not think such a diagnosis fits with the history at all. He certainly has not the ordinary type of Bright's disease. The examination also shows that the urine is very concentrated; but it is not the high specific gravity due to diabetes unless he has some sugar in his urine, and he does not show any. There is a large trace of albumin. I think we can disregard the leukocytes in the sediment. Twenty-five leukocytes per high power field might be very significant; but certainly it could not explain the rest of the clinical picture. It is rather a strange picture as we go along.

Fifty per cent is probably the right renal function, because the higher figure should be taken. It may be concentrated, and 50 per cent is an over-reading. That is a good renal function by the phenolsulphonephthalein test. The kidneys are not the fundamental element in this particular case. We know they are functioning improperly because there is a great deal of albumin. Nephritis could explain most of the physical findings in this case, but it will not cause night sweats, afternoon fever, or adenopathy.

I think amyloid disease can give a somewhat similar picture. If nephrosis is the cause of this particular picture we should find other things. We should find low serum protein and serum albumin. We usually have a low metabolic rate. I wonder whether we shall find any of these things here.

The finding of myeloblasts injects another element of doubt in this diagnosis, if those were real myeloblasts. He has a moderate leukocytosis which cannot be overlooked. He has an anemia which is moderately severe, secondary in type. The rest of the blood shows some stimulation of the bone marrow. If these are myeloblasts, if the marrow is very much stimulated, we should have more than 65 per cent polymorphonuclear cells, more eosinophiles or basophiles. I am wondering whether they were myeloblasts or whether they were "unclassified" cells. We think he has glandular involvement

in the abdomen. It is conceivable that he may have some type of leukemia, not myeloid leukemia. I should think he did not have it. Besides, I believe five to seven years, if we can believe his story, is longer than the usual course of leukemia. The red cells appeared normal, and with the exception of the myeloblasts they do not speak much of abnormal white cells.

The non-protein nitrogen is perfectly normal, and is another point against chronic nephritis.

This man has not diabetes from any evidence we have. I think that can be completely forgotten. As he has amyloid disease of his pancreas any extra strain would make him show a little sugar. I am inclined to disregard the glycosuria.

I do not know anything about the albumin and globulin ratio. In cases of so-called nephrosis we should have a very much lower serum protein than 5.27 per cent.

The guaiac test on the stools fits in with the diarrhea. I think that is entirely consistent with tuberculosis, tuberculous colitis or considerable glandular involvement.

The basal metabolic rate is probably perfectly normal as far as this patient is concerned. He has a temperature up to 101.4°, and one might expect a slight elevation of metabolic rate. A rate of + 18 is one more point against nephrosis. So far there is nothing in the picture against an advanced glandular tuberculosis.

Pulmonary tuberculosis is certainly out of the picture so far as the X-ray is concerned. Tabes mesenterica as a rule shows a pulmonary lesion, but I do not think it is necessary for such a diagnosis.

The patient was a sick boy and was not able to stand, and the X-ray finding of the high diaphragm may mean merely distention from fluid in his belly. I should think that those X-rays were simply inconclusive. A moderate degree of colitis may or may not give striking X-ray changes. In cases of moderate ulceration of the bowel the results of barium enemas are sometimes very unsatisfactory in demonstrating real colitis.

The temperature was certainly an up and down one. I do not see why it does not fit in perfectly well with a diagnosis of tuberculosis. It is surprising that the temperature is not a little higher, but there was a swing of nearly four degrees. That is a pretty good swing.

I take it that one reason for the disappearance of the edema of the legs was that he was placed on his back in bed.

A dry tap does not mean there was no fluid in the abdomen. It simply means they did not get it. "Abdomen tympanitic" is entirely consistent with glandular disease. He may have had fluid colonic contents instead of ascites.

Alpine lamp treatment is the type of treatment one would give in glandular tuberculosis. Although it can be used for other diseases, that is the disease in which it is most logically employed.

I take it that there was an exploratory operation. The surgeons were looking for a disease of the glands. So far there is no reason to think that it might not have been tuberculous. However, a gland was removed, and we have to consider that there may have been glandular disease other than tuberculosis and probably not syphilis. I suppose a chronic, very rare leukemia might be considered here, or Hodgkin's disease. I have not mentioned the latter before. It could give this picture with abdominal glands. No splenic enlargement has been mentioned. There were hard glands in the groin. He may have Hodgkin's disease. It is rather a long course for Hodgkin's disease in a patient of nineteen years. Usually it is a matter of two or three years' duration rather than five or six. Patients with Hodgkin's can have night sweats. They may give a very striking picture of long duration. We have to consider tuberculosis of the glands, particularly in the mesentery, with a very good possibility of amyloid in the kidneys or Hodgkin's disease with involvement of the kidneys.

Nothing was done at operation except to make a diagnosis. Possibly they found a lot of glands and decided that there was nothing more to be done by surgical methods.

I think the nosebleed occurring after operation is of no importance.

The ankles swelled again because the old cause of the edema is still there.

X-ray treatment was given. They thought he had Hodgkin's disease or, as a poorer bet, leukemia.

Hodgkin's disease is still to be considered. This is not the usual blood smear of Hodgkin's disease. One would expect to find not quite so high a leukocyte count and a very much higher polymorphonuclear count. Increased platelets are a characteristic finding of late Hodgkin's disease. The 25,000 leukocyte count is against tuberculosis. I think that is the one thing in the picture so far against tuberculosis. There was constant leukocytosis, therefore I am inclined to consider very definitely the possibility of Hodgkin's disease here, with tuberculosis a close second. The smear of Hodgkin's disease is not diagnostic, but it runs fairly true to form.

The pleural effusion may have been due to a subdiaphragmatic process around the liver, or to some involvement of the pleura just before he died.

The outstanding features of this disease are its chronic nature, fever, night sweats, cachexia, adenopathy, ascites, albuminuria, subsequent edema and finally diarrhea. We have to assume some renal damage here or else he would not have so marked an albuminuria. I think the diagnosis is one of glandular disease, lying between tuberculous glands with amyloid degen-

eration of the kidneys or Hodgkin's disease of longer duration than usual, of the Pel-Ebstein type, with intermittent rises in temperature, probably with involvement of the mesenteric glands and the kidney structure. I am inclined to lean toward Hodgkin's disease, because of the leukocytosis and because of the fact that he received radiotherapy.

PRE-OPERATIVE DIAGNOSIS

Tuberculosis of the peritoneum.

OPERATION

Under spinal anesthesia the abdomen was opened through a right paramedian incision. It contained a large amount of clear fluid. There was no evidence of tuberculosis of the parietal or visceral peritoneum. In the mesentery of the jejunum there was a rounded tumor as large as a base ball, representing without doubt an enlargement of the lymphatic glands, as other glands in the mesentery lower down were also enlarged. These glands were soft and a little redder than normal, evidently a lymphoblastoma. The lymphoid tissue of the gut itself was not involved. One gland was taken for examination.

PATHOLOGIC REPORT

A soft lymph node the size of a Lima bean, with a smooth pale red homogeneous surface on section.

On microscopic examination the histologic structure is preserved. The sinuses are distended with serum and contain numerous polynuclears and large mononuclear leukocytes. There is a moderate degree of activity in the follicles. There is no evidence of neoplasm or tuberculosis.

Chronic inflammation.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Abdominal lymphadenopathy, probably lymphadenoma.

DR. CHESTER M. JONES'S DIAGNOSIS

Hodgkin's disease of the mesenteric lymph glands, with associated infiltration of the kidneys; or abdominal glandular tuberculosis with amyloid degeneration of the kidneys.

ANATOMIC DIAGNOSES

Amyloid nephrosis.
Amyloid degeneration of the liver, spleen, accessory spleen and adrenal.

DR. TRACY B. MALLORY: The autopsy will explain part of the picture, I am afraid perhaps only the smaller part. The autopsy was done at the patient's home. Three rather striking things were found. The liver, spleen, and kidneys were all unusually large, pale and firm. There was also a tumor mass about the size of a small orange found in the root of the mesentery, and around that were a number of smaller glands from 1 to 1.5 centimeters in size, one of which had been removed at the exploratory operation for biopsy. The gross appearance of the organs suggested very strongly amyloid disease. That was confirmed by histologic examination. Typical staining reactions of chocolate brown with iodine and bright pink with gentian violet were obtained. The lymph nodes throughout the body were enlarged and all of them showed a marked degree of amyloid infiltration. The big mass in the mesentery seemed at first to be tumor, but sections of it showed such complete replacement with amyloid that very little could be definitely identified. We could however make out a certain number of hyalin masses suggesting the changes which amyloid produces in splenic corpuscles, and also trabeculae of fibrous tissue very characteristic of the spleen. So our eventual opinion was that the tumor was an aberrant spleen. These can occur anywhere in the abdominal cavity although usually it is somewhere near the left upper quadrant. Nowhere in the body could we find any definite evidence of either Hodgkin's disease or tuberculosis or any other infectious disease. What the cause of his amyloid was, what the cause of his continued fever, we have no explanation.

The case seemed quite interesting, particularly because amyloid disease used to be one of the commonest findings at the autopsy table. In all large hospitals thirty years ago a bottle of iodine was kept in the laboratory to test out almost every liver as it came along, with the expectation of finding at least one in twenty that would give a definite amyloid reaction. This is the first case of at all marked amyloid we have had in four years at this hospital. It is a disease which so far as is indicated by the material which we have a chance to see seems to be disappearing as fast as chlorosis, unquestionably common thirty years ago, but the greatest of rarities now.

The kidneys showed the usual picture of marked amyloid infiltration of all the glomeruli and also very marked hyalin droplet degeneration of the tubular cells, typical of what we call nephrosis. Most of the German writers prefer to class amyloid as a type of nephrosis. The reason for that seems fairly obvious from the symptoms in this case.

DR. JONES: I wish that I had stuck to my diagnosis of tuberculosis and amyloid. One can get a very striking degree of albuminuria with that. I have never seen such a case, but it seems to me fair to assume that he had chronic infection as evidenced by his night sweats and fever over a long period of time, with resulting amyloid disease. Tuberculosis I suppose would be the most logical cause.

DR. MALLORY: It most certainly would be.

A STUDENT: Would Hodgkin's present such a picture in the abdomen as this boy presented?

DR. JONES: Abdominal Hodgkin's can do this.

DR. MALLORY: You can also occasionally get amyloid disease with Hodgkin's, although it is rare.

DR. JONES: You can get sufficient infiltration of the kidneys so that you may have albuminuria.

A STUDENT: What was the basis of X-ray treatment?

DR. JONES: I presume they thought there was Hodgkin's disease. They do not use X-ray with tuberculosis.

There was fluid in the abdomen, which shows that a dry tap does not mean that there is no fluid present.

DR. MALLORY: That biopsy specimen showed amyloid which was discovered later, but not noted at the time.

CASE 15432

A SUITABLE CASE FOR TREATMENT WITH QUINIDINE SULPHATE

CARDIAC CLINIC OF THE MEDICAL DEPARTMENT

First admission. A Canadian meat cutter fifty-two years old entered January 9 complaining of dyspnea on exertion and epigastric discomfort. After examination in the Out-Patient Department January 8 the possibility of perforating gastric ulcer was suggested.

He had always been in very good health until eight weeks before admission. Then he got a bad cold with severe cough, dry at first. In two weeks the cold was gone, but the cough and dyspnea continued, becoming worse. The cough was more severe after exertion, at night, and on getting up. With the onset of the cold he began to have dyspnea on exertion. He continued his work in an ice chamber in the market, doing heavy lifting. He occasionally had dyspneic attacks while sitting still or at night, coming and going suddenly and lasting only about two minutes. The epigastric discomfort came on especially after exertion, not a definite pain. It was intensified by an unusually heavy meal, but was not definitely associated with meals. He felt as if he were distended with gas, but had no belching. For three weeks he had not dared to eat heartily. For two weeks the cough had been productive of thick yellowish sputum, most of it in the morning on getting up, as much as a cupful and a half. Since the onset his bowels had been constipated. He had lost about seven pounds in the past eight weeks. For years the circulation had been poor in his hands and feet. They were often blue and cold. He associated this with his occupation.

There is no history of any familial disease.

He had always had winter cough with thick yellow sputum. Twenty-five years before admission he had gonorrhea. He denied symptoms of syphilis. For forty years he had taken about half a pint of liquor a week. He seldom was intoxicated.

Clinical examination showed a well nourished man with slight dyspnea and some cyanosis. There was early arcus senilis. The chest was broad and deep, hyperresonant, with shallow excursion of the diaphragm and coarse râles in both bases. The heart was slightly enlarged. The apex impulse was ten centimeters to the left of the midsternum. The heart was absolutely irregular in rate and rhythm. The apex rate was 130. The sounds were of fair quality. The peripheral vessels showed evidence of moderate arteriosclerosis. Blood pressure 128/98 mm. to 105/85 mm. January 10 an electrocardiogram showed auricular fibrillation, rate 130, flat T_2 wave. The lower abdomen was soft. There was slight spasm of the upper portion, with definite tenderness over the liver, especially the left margin. Both legs showed marked varicosities. The pupillary reactions and other reflexes were normal.

Urine: 32 to 75 ounces, specific gravity 1.014, 4 to 12 leukocytes per high power field, renal function 25 per cent. Blood normal. Wassermann negative January 8 and 21. Non-protein nitrogen 39.6 milligrams.

X-ray examination showed the heart shadow considerably enlarged both to the right and to the left. The supracardiac shadow was not increased in width (see Figure 1). The outline suggested tortuosity. The enlargement was considerably more than is usually seen in the presence of hypertension alone.

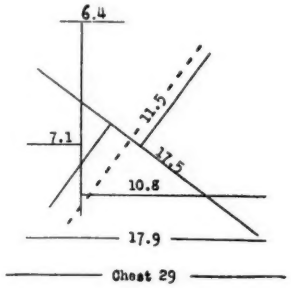

FIG. 1.

Temperature 96.5° to 99.5°. Radial pulse 120 to 48, with a pulse deficit of 8 to 50. Respirations 20 to 35.

Under calcium chloride 2.5 grains six times a day, rest in bed and a soft solid diet the patient made good improvement. January 19 a heart consultant found the ventricular rate 120 or more, evidence that although rest in bed and calcium chloride as a diuretic had been of considerable value in the patient's improvement there had been no effect on the fibrillation or the heart rate. January 22 X-ray examination showed that the shape of the heart had the same general appearance, but the size was consider-

ably diminished. (See Figure 2.) On January 23 he was given 12 grains of digitalis, on January 24 the same amount and on the 25th one pill of a grain and a half. On the same day he received two doses of three grains each of quinidine sulphate. January 26 another electrocardiogram showed auricular fibrillation, rate 70. On January 26 and 27 he was given five doses of quinidine of six grains each. The morning of the 27th the heart was regular after he had taken 35 grains of quinidine. After the heart returned to normal rhythm the dose was reduced to three grains twice a day. January 28 he felt very much better. The epigastric pain had entirely disappeared. A third electrocardiogram showed normal rhythm, rate 56, P-R interval 0.2 seconds, diphasic T_2. January 30 he was discharged.

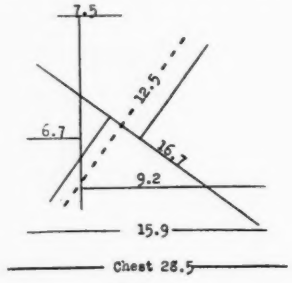

FIG. 2.

History of interval. The patient was not seen in the Out-Patient Department until July 15, four years later, when he returned because he was troubled by gas and dyspnea. He had been working hard at his old occupation, which required the lifting and carrying of heavy weights. About July 12, when he was lifting 100 pounds of sugar to his shoulder, his heart suddenly became irregular. September 5 he was seen in the Cardiac Clinic and the diagnosis of recurrent auricular fibrillation was confirmed. He was readmitted to the wards for another trial of quinidine sulphate. He had been digitalized on a visit to the medical Out-Patient Department in July.

Second admission, September 6, four years and seven months after his discharge.

Clinical examination showed a florid, healthy looking man who had dyspnea on relatively slight exertion. Teeth, three stumps. Slight pyorrhea. Tonsils large. Fine râles at both bases, also coarse râles at the right base with increased breath sounds and slightly muffled vocal fremitus. Apex impulse of the heart not seen or felt. The heart appeared markedly enlarged. Percussion poor because of high gastric tympany. Left border of dullness 10¾ centimeters to the left of midsternum, right border 3 centimeters, supracardiac dullness 5.5 centimeters. Heart absolutely irregular. No pulse deficit. Sounds of fair quality. A systolic murmur, loudest at the fourth space. Radials palpable. Blood pressure 146/102. An electrocardiogram September 7 showed auricular fibrillation, partial auriculoventricular block, ventricular rate 40, inverted T_1 and T_2. Vital capacity September 7 and 9; 2900 to 3050.

Urine, renal function and blood normal. Hinton negative.

Temperature and respirations normal. Pulse 40 to 79.

X-ray examination showed that the heart shadow had changed somewhat in shape since the last observation. There was still considerable increase in size. (See Figure 3.) The increase was now largely to the left in the region of the ventricle. The aorta was tortuous and prominent in the region of the knob.

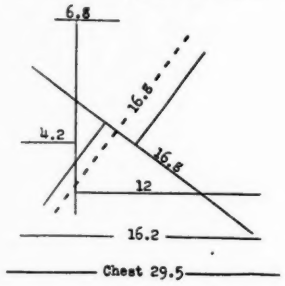

FIG. 3.

Quinidine was started on September 8. After 18 grains the pulse deficit increased twenty beats. After 30 grains normal cardiac rhythm was restored except for occasional premature beats. The quinidine was reduced to six grains a day on September 9 and to three grains a day two days later because of some headache and vomiting. It was omitted entirely on the 15th. He was discharged on the 17th with normal rhythm.

On October 3, two weeks later, he came to the Cardiac Clinic, where the cardiac examination showed a regular rhythm at a rate of 58. He was advised to use six grains of quinidine a day if he felt his heart to be irregular or after suffering from any infection or intestinal disturbance.

He was last seen January 30, four months later, in the Cardiac Clinic. His heart was regular at 64. The blood pressure was 165 systolic and 100 diastolic. His only complaint at that time was a transient precordial pain not related to exertion.

Discussion

BY HOWARD B. SPRAGUE, M.D.

This case is presented because it shows the type of patient who is suitable for a trial of quinidine sulphate to restore normal rhythm in auricular fibrillation. This man had little evidence of cardiac disease. There was a certain amount of arteriosclerosis and hypertension, but no marked congestive heart failure either during observation at the hospital or according to the past history. There was also no evidence of chronic valvular disease or very marked enlargement of the heart, and the auricular fibrillation had been of short duration.

Sudden severe exertion on two occasions had resulted in the initiation of abnormal cardiac rhythm. The chief complaints from this were palpitation and uncomfortable chest sensations rather than shortness of breath. Restoration of normal rhythm was quite easily accomplished with relatively small doses of quinidine sulphate and made it possible for him to continue laborious work for over four years between the attacks of auricular fibrillation. The circulation was doubtless more efficient during normal rhythm than during auricular fibrillation, even though the heart rate could have been controlled by digitalization.

It is probable however that auricular fibrillation will recur more easily as time goes on. He should be given a daily ration of six grains of quinidine during periods of mild infection or of particular strain in an attempt to put off the recurrence of the irregular rhythm.

INTERPRETATION OF X-RAY, SECOND ADMISSION

Arteriosclerosis. Hypertrophy of the left side of the heart and dilatation of the auricles.

DIAGNOSES

Hypertensive and arteriosclerotic heart disease.
Auricular fibrillation.
Congestive failure.
Angina pectoris?

METHODS OF DETECTING THE PRESENCE OF CARBON

Japanese waltzing mice have been found to be more responsive to concentrations of the deadly carbon monoxide than canaries, long used by the United States Bureau of Mines, Department of Commerce, for this purpose in connection with mine rescue operations. On the other hand, guinea pigs have been found to be unsuited for detecting dangerous amounts of carbon monoxide.

The use of small animals depends on the fact that, due to their high metabolic rate and oxygen demand accompanied by high respiratory exchange and rate of circulation, they respond more quickly than man to a given concentration of gas. From a practical viewpoint this allows a margin of time between observable serious effects to the animal and serious effects to man, during which period man can return to fresh air or employ means of respiratory protection. It should be remembered, however, that during the time the animal is incurring a detectable degree of poisoning, man is also absorbing some carbon monoxide, and in some cases may attain a very discomforting degree of poisoning, especially if the particular animal is more tolerant than the average. The margin of time between effects in the animals and in men is not very wide and experience with canaries has shown that an occasional specimen may be tolerant enough to fail to exhibit symptoms before serious effects are incurred in men.

It is also known that carbon monoxide poisoning is augmented by physical activity. The latter further increases both the demand for oxygen, and the respiratory exchange and circulation of the blood. For this reason, when using animals for detecting carbon monoxide, it is endeavored to keep them active and at the same time minimize activity on the part of the men, thereby tending to increase the sensitivity and widen the time between observable effects in the birds and effects in man. This may be accomplished to a certain extent with canaries but ordinary mice and white mice tend to become more inactive and huddle in the corner of the cage thereby reducing their sensitivity.

In view of these facts, it was thought that due to their almost incessant activity, Japanese waltzing mice might be more susceptible to carbon monoxide than either canaries.

In comparative tests with canaries made at the Pittsburgh Experiment Station of the Bureau of Mines, it was found that the Japanese waltzing mice showed signs of response to carbon monoxide in advance of signs in the birds. The signs were first manifested in a weakness of the hind limbs and falling, while performing the erratic movements; followed by a slowing down of movements and later inability to perform the movements. The sensitivity to the gas increased with the degree of activity of the various specimens used. The recovery after exposure was rapid, but a few minutes being required for apparent recovery from a degree of poisoning causing unconsciousness.

In the course of other experimental work conducted at the Pittsburgh Experiment Station, it has been strikingly shown that guinea pigs would not be suitable for detecting dangerous amounts of carbon monoxide. Animals of this species were exposed to air containing 0.17 to 0.25 per cent. carbon monoxide and exhibited only very slight, and not objectively distinct symptoms, at the end of 18 hours, slight to moderate symptoms at the end of 24 hours, and the majority were able to move around, though unsteadily, at the end of 34 hours. The same amount of carbon monoxide in air would cause unconsciousness in a man doing but moderate work as walking, in 30 minutes to one hour, and death in about four to six hours. When encountering potentially dangerous atmospheres the Bureau of Mines usually employs mechanical devices as the flame safety lamp for detecting atmospheres deficient in oxygen, and the hoolamite carbon monoxide detector for detecting carbon monoxide, but where small animals are required only canaries have been used.—*Department of Commerce, Washington.*

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SOCIALIZATION OF MEDICINE

COMMUNISM is sometimes thought of as a scheme in which those who will not work are supported by those who will. Why not? Some people naturally like to exert themselves, others do not. Socialism is regarded as a somewhat less extreme form of a scheme essentially the same in principle. Neither of these formulas actually fits the facts. In some form, the theory demands, to prevent undue antisocial activity, the control of all of us by every body else. Its philosophical justification lies in the ultimate interrelatedness of humanity (as well as the rest of the universe). It breaks down, however, because it has not secured the backing of common sense.

But there is an ineradicable tendency toward socialism, and the socialization of medicine is a transition, which going on before our eyes, brings to some of us satisfaction, to others consternation. In some respects, the conservatives say, it may go too far. But there is comfort for the uncomfortable in the fact that the tendency toward individualism is just as ineradicable, and suppressed at one point it will break out at another. Neither the individualist nor the socialist can reign supreme for long.

It is in the field of preventive medicine that the socialization of medicine has proceeded most rapidly in recent years, and a factor in the spread of preventive medicine is the public health nurse. This is at least one reason why Miss Beard's studies[1] supplementary in some respects to Miss Gardner's *Public Health Nursing*[2] should be read by every physician interested in the broader aspects of his work.

The studies are based largely on first hand knowledge. The book reviews conditions in several parts of Europe as well as in this country, considers some current problems of public health nursing administration, and presents an interesting look into the future in the closing chapter. Whether the author has the gift of prophecy or of fancy, time alone will tell.

But what is brought out by much of the testimony of Miss Beard, confirmed from other sources, is that while the nurse in public health may have been a factor in the socialization of medicine, at the same time she has been, and is and will be, an important factor in the individualization of health. This second result, in the production of which the nurse plays only a part, no physician can regret and the problem set (not solved) by the book is: How can it be done better and more efficiently. This is a problem for every physician, to which each, in his restricted sphere, can make some contribution, in practice if not in theory.

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1 M. Beard: The Nurse in Public Health. Harper. 1929.
2 M. S. Gardner: Public Health Nursing. Macmillan.

RESUMPTION OF THE CAMPAIGN FOR RAISING MONEY FOR THE BOSTON MEDICAL LIBRARY AND THE MASSACHUSETTS MEDICAL SOCIETY

THE committees for raising money for the additions to the Medical Library and the home for the Massachusetts Medical Society are preparing for the renewal of the Campaign conducted last winter and spring.

A good beginning was made in the first drive which was concentrated on physicians and their immediate friends. There were encouraging responses on the part of a large number of doctors but the numerical response was less than seemed probable at first. A considerable proportion of the doctors in and about Boston evidently did not feel that degree of personal responsibility which would otherwise have led to more general contributions. In this movement one of the essential features is the evidence of unanimous endorsement through such contributions as can be afforded and especially by the demonstration of moral support in presenting the appeal to those who would be likely to respond.

Of course every community has its own standards of responsibility toward common needs and Boston can and should stand as a leader in the support of medical institutions. Our Library stands high among the great medical libraries of the world and civic pride alone should inspire the profession to maintain it to the highest degree of usefulness. To do this every doctor in this section who is interested in scientific medicine is under obligation to use his influence in augmenting the work of those who are devoting their time to this project. There should be evidence of co-öperation on the part of at least ninety per cent. of the membership of the Massachusetts Medical Society. Will the Campaign fail to meet its objective because of the inertia of the profession?

MORE CARE IN CRITICISM OF HOSPITALS IN ORDER

According to one's mental habits and training public expressions of opinion may be helpful or damaging when dealing with subjects concerning which the public is interested.

Physicians as a class should understand that reactions of the average person to statements may arouse misunderstanding or resentment. The old admonition to cultivate the wisdom of the serpent and yet be as harmless as doves seems to apply at the present time when doctors unburden their minds on subjects which appeal to the laity. It is common knowledge that the report of the saying of an eminent physician with respect to the proportion of errors in diagnoses has been seized upon and quoted by many in arguments designed to confute the pleas of doctors.

It may be that the statement has not been correctly reported and too it may be that serious abuses exist in other parts of the country more than in the region with which we are familiar; and further that the author of the statement has facts at his command justifying his accusation. However, those who are engaged in the work of trying to convince the public of the duty to support hospitals may wish that if abuses exist the effort to correct them should be inaugurated without public denunciation of a general character. We believe in specific statements of wrong doing but deplore sweeping charges of objectionable practice without corroborative facts. Since the American College of Surgeons places its approval on a considerable proportion of the hospitals of the country it may be fair to assume that the exploiting of patients may not be common in the approved group of institutions.

We respectfully suggest that unless it seems to be necessary in certain cases physicians should be careful to avoid criticising hospitals or making other statements which may not have specific application. By all means doctors should try to bring about reforms when indicated, but it is not good policy to furnish unsympathetic persons with arguments which will do harm.

THIS WEEK'S ISSUE

Contains articles by the following named authors:

Stevens, Neil C. B.A., M.D. Columbia University College of Physicians and Surgeons 1915. Instructor in Medicine, Cornell Medical School; Attending Physician, Cornell Clinic and North County Commission Hospital, Glen Cove. His subject is: "Endocrine and Fatigue Headaches." Page 801. Address: Glen Cove, Long Island.

Allen, Frederick M. A.B., M.D. University of California 1907. Formerly Research Fellow, Harvard Medical School and Associate in Medicine, Rockefeller Institute, New York City. Now Director of Physiatric Institute, Morristown, N. J. His subject is: "Observations on Sweating." Page 806. Address: The Physiatric Institute, Morristown, N. J.

Bauer, Walter. B.S., M.D. University of Michigan 1927. Instructor, Harvard Medical School; Assistant Physician, Massachusetts General Hospital. Address: Massachusetts General Hospital. Associated with him is:

Marble, Alexander. A.B., A.M., M.D. Harvard 1927. Medical Resident, Massachusetts General Hospital. Address: Massachusetts General Hospital. Their subject is: "Preliminary Note on the Mode of Action of Irradiated Ergosterol." Page 809.

Arnold, Lloyd. A.B., A.M., M.D. Vanderbilt 1918. Bacteriologist, State Department Public Health. His subject is: "Entero-Intoxication." Page 811. Address: 1817 W. Polk Street, Chicago, Illinois.

Ulrich, Helmuth. M.D. Boston University School of Medicine 1911. Associate Professor of Clinical Pathology, Boston University Medical School; Chief of Diabetic Service, Massachusetts Homeopathic Hospital; Research Associate, Evans Memorial for Clinical Research and Preventive Medicine. His subject is: "Synthalin-B In Diabetes Mellitus." Page 814. Address: 416 Marlborough Street, Boston.

Horlick, S. Seymour. B.B.A., M.D. Tufts College Medical School 1928. Out-Patient Department, Beth Israel Hospital. Address: 1687 Commonwealth Avenue, Boston, Mass. Associated with him is:

Bicknell, Ralph E. M.D. Harvard 1900. Chief of Medical Service, Lynn General Hospital. Address: Swampscott, Mass. Their subject is: "Trichiniasis with Widespread Infestation of Many Tissues." Page 816.

HAWES, JOHN B. 2ND. A.B., M.D. Harvard 1903. President, Boston Tuberculosis Association. Address: 11 Marlborough Street, Boston. Associated with him is:

STONE, MOSES J. M.D. Tufts Medical School 1921. Formerly on the Staff of Massachusetts State Infirmary, Tuberculosis Department Rutland State Sanatorium and Westfield State Sanatorium. Now examiner for the Boston Health Department of Tuberculosis Clinics; Physician at the Chest Clinic, Massachusetts Homeopathic Hospital Out-Patient Department; Assistant in Medicine, Beth Israel Hospital and Boston University Medical School. Address: 11 Marlborough Street, Boston. Their subject is: "Progress in Tuberculosis 1928-1929." Page 820.

DALTON, CHARLES F. M.D. University of Vermont College of Medicine 1903. Secretary and Executive Officer Vermont Department of Public Health since 1912. Professor of Hygiene and Preventive Medicine University of Vermont College of Medicine. Formerly President, Conference of State and Provincial Health Authorities of North America and Vermont State Medical Society. His subject is: "Communicable Disease Problem." Page 829. Address: Burlington, Vermont.

MISCELLANY

DR. CUSHING SPEAKER AT JOHNS HOPKINS

PROTESTS LIBRARY BOOKS LEFT UNDISTURBED

Special Dispatch to the *Globe*

BALTIMORE, October 17 — Expressing the hope that medicine would in the future assemble "all her foster children" about her in the great Welch Medical Library of Johns Hopkins University, Dr. Harvey Cushing, professor of surgery in Harvard University, in an address at the dedicatory exercises of the library today said that too often physicians were too busy practicing medicine to know anything of the history of their science.

By Medicine's foster children, Dr. Cushing made it clear that he referred to all the divisions and subdivisions of the science which have grown up in the modern aspects of medicine. He said that frequently practitioners of medicine were not familiar with the history of that science.

Dr. William H. Welch, in whose honor the splendid building was erected, was surrounded on the platform by many notable savants in medicine and surgery, some of his former students including Dr. William T. Councilman, professor of pathology at Harvard University; Dr. Simon Flexner, Rockefeller Institute, New York, and Dr. E. L. Opie, of the University of Pennsylvania, director of the Henry Phipps Institute of Pennsylvania.

Dr. Cushing of Harvard, a former member of the Hopkins faculty and the author of "The Life of Sir William Osler," who was the first professor of medicine at the Hopkins Medical School, referred at the beginning of his address to libraries in which the books remained undisturbed on their shelves and expressed the hope that in this library the books would be alive to those who were privileged to use them.

Beside Dr. Welch sat the whitehaired Dr. Karl Sudhoff, professor emeritus of the history of medicine of the University of Leipzig and the greatest authority on the history of medicine in the world.

Others present were:

Dr. J. C. Merriam, president of the Carnegie Institution; Dr. E. B. Krumbhaar, Philadelphia; Dr. Henry Pritchett, president of the Carnegie Foundation; Dr. M. G. Seeling, St. Louis, medical historian; Dr. Florence R. Sabin, Rockefeller Institute, New York; Professor Otto Warburg of Kaiser Wilhelm Institute, and former professor of histology at Johns Hopkins Medical School, and Senator and Mrs. Frederick C. Walcott, Norfolk, Conn. Senator Walcott is a nephew of Dr. Welch.

TWO FELLOWSHIPS FOR THE WILMER OPHTHALMOLOGICAL INSTITUTE

Two research fellowships for the Wilmer Ophthalmological Institute at Johns Hopkins University were announced at the dedicatory exercises of the Institute October 15, 1929. One was donated by Andrew W. Mellon and his brother and the other by Adolph Lewisohn of New York. Each carries a gift of $30,000.

Among the eminent men present at the exercises were Dr. George E. Vincent and Herbert L. Satterlee.

The Rockefeller Foundation donated $1,500,000 to the $4,000,000 fund for the Wilmer Institute.

Mrs. Henry Breckenridge is credited with a large share of the influences which have made possible the endowment of the Institute.

RHODE ISLAND MORTALITY STATISTICS: 1928

Washington, D. C., October 4, 1929.—The Department of Commerce announces that the 1928 death rate for Rhode Island was 1,172.3 per 100,000 population as compared with 1,121.7 in 1927.

Increases in rates (per 100,000 population) from those of the preceding year were from the following principal causes: diseases of the heart (211.4 to 232.5), cancer (110.2 to 118.2), nephritis (104.4 to 112.2), and diabetes mellitus (20.6 to 25.4). Increases were shown also for pneumonia, all forms (85.2 to 98.5), measles (0.1 to 9.8), influenza (12.5 to 14.2), whooping cough (5.7 to 6.4), and meningococcus meningitis (0.7 to 1.3).

The death rate from all accidental causes increased from 61.9 to 67.6, the individual types of accidents, showing the greatest increases, being automobile accidents, excluding collisions with railroad trains and street cars (18.6 to 21.5) and machinery accidents (0.9 to 1.5).

Significant among the decreases in rates from 1927 to 1928 were those from the following principal causes: tuberculosis, all forms (73.3 to 68.7) and cerebral hemorrhage and softening (107.7 to 104.1). Other decreases were shown for congenital malformations and diseases of early infancy (74.0 to 63.8), diphtheria (8.7 to 7.0), rheumatism (4.4 to 2.7), acute anterior poliomyelitis (infantile paralysis) (1.6 to 0.6), diarrhea and enteritis, under 2 years (13.6 to

Cause of Death	Deaths in Rhode Island			
	Number		Rate per 100,000 estimated population	
	1928	1927	1928	1927
ALL causes 1/	8,394	7,897	1,172.3	1,121.7
Typhoid and paratyphoid fever	10	4	1.4	0.6
Malaria	-	-	-	-
Smallpox	-	-	-	-
Measles	70	1	9.8	0.1
Scarlet fever	16	15	2.2	2.1
Whooping cough	46	40	6.4	5.7
Diphtheria	50	61	7.0	8.7
Influenza	102	88	14.2	12.5
Dysentery	1	4	0.1	0.6
Erysipelas	19	19	2.7	2.7
Acute anterior poliomyelitis	4	11	0.6	1.6
Lethargic encephalitis	4	7	0.6	1.0
Meningococcous meningitis	9	5	1.3	0.7
Tuberculosis (all forms)	492	516	68.7	73.3
Of the respiratory system	390	439	54.5	62.4
Of the meninges, central nervous system	42	45	5.9	6.4
Other forms	60	32	8.4	4.5
Syphilis 2/	80	77	11.2	10.9
Cancer and other malignant tumors	846	776	118.2	110.2
Rheumatism	19	31	2.7	4.4
Pellagra	1	2	0.1	0.3
Diabetes mellitus	182	145	25.4	20.6
Meningitis (nonepidemic)	26	28	3.6	4.0
Cerebral hemorrhage and softening	745	758	104.1	107.7
Paralysis without specified cause	7	13	1.0	1.8
Diseases of the heart	1,665	1,488	232.5	211.4
Diseases of the arteries, atheroma, aneurysm, etc.	187	174	26.1	24.7
Bronchitis	49	50	6.8	7.1
Pneumonia (all forms)	705	600	98.5	85.2
Respiratory diseases other than bronchitis and pneumonia (all forms)	48	56	6.7	8.0
Diarrhea and enteritis	116	127	16.2	18.0
Diarrhea and enteritis (under 2 years)	91	96	12.7	13.6
Diarrhea and enteritis (2 years and over)	25	31	3.5	4.4
Appendicitis and typhlitis	93	90	13.0	12.8
Hernia, intestinal obstruction	78	69	10.9	9.8
Cirrhosis of the liver	37	39	5.2	5.5
Nephritis	803	735	112.2	104.4
Puerperal septicemia	23	29	3.2	4.1
Puerperal causes other than puerperal septicemia	56	59	7.8	8.4
Congenital malformations and diseases of early infancy	457	521	63.8	74.0
Suicide	67	65	9.4	9.2
Homicide	18	18	2.5	2.6
Accidental and unspecified external causes	484	436	67.6	61.9
Burns (conflagration excepted)	39	36	5.4	5.1
Accidental drowning	50	43	7.0	6.1
Accidental shooting	3	2	0.4	0.3
Accidental falls	110	127	15.4	18.0
Mine accidents	-	-	-	-
Machinery accidents	11	6	1.5	0.9
Railroad accidents	12	15	1.7	2.1
Collision with automobile	2	-	0.3	-
Other railroad accidents	10	15	1.4	2.1
Street-car accidents	19	12	2.7	1.7
Collision with automobile	6	3	0.8	0.4
Other street-car accidents	13	9	1.8	1.3
Automobile accidents (excluding collision with railroad trains and street cars)	154	131	21.5	18.6
Injuries by vehicles other than railroad trains, street cars, and automobiles 3/	12	3	1.7	0.4
Excessive heat (burns excepted)	6	1	0.8	0.1
Other external causes	68	60	9.5	8.5
All other defined causes	748	694	104.2	98.6
Unknown or ill-defined causes	33	46	4.6	6.5

1/ Exclusive of stillbirths.
2/ Includes tabes dorsalis (locomotor ataxia) and general paralysis of the insane.
3/ Includes airplane, balloon, and motor-cycle accidents.

12.7), and lethargic encephalitis (sleeping sickness) (1.0 to 0.6). The death rate from accidental falls decreased from 18.0 to 15.4.

The estimated population in 1928 was 716,000 and in 1927 was 704,000.—*Department of Commerce, Bureau of the Census, Washington.*

THE MIDDLESEX HEALTH ASSOCIATION

The annual meeting and election of officers of the Southern Middlesex Health Association, Inc., was held at Medford, October 16, 1929. The annual report of the executive secretary, Miss Mary C. Hoisington, was read and the financial report of Clarence C. White of the Reading National Bank, chairman of the finance committee.

The election of officers resulted in the re-election of Dr. Carl C. MacCorison of the North Reading State Sanatorium, president. Dr. Wilfred L. McKenzie of the Children's Hospital, Boston, first vice-president and William J. Bell of Somerville, second vice-president.

E. Earl Blakley of the Medford Trust Company was elected treasurer and Mrs. Claude E. Patch of Stoneham, secretary. The executive committee is: Arthur W. Coolidge of Reading, Mrs. Elisha Fowler of Reading, Mrs. C. C. Hunt of Everett, Dr. Charles A. McPherson of Medford, James J. Quinn of Winchester, Winthrop G. Rockwell of Watertown, Almon E. Richardson of Melrose, Dr. Therese B. Thomas of Arlington, Clarence C. White of Reading, and Frank L. Scott of Belmont.

Dr. Charles F. Wilinsky, director of the Beth Israel Hospital, spoke on "Developing Importance of Child Health Activities."

UNDULANT FEVER IS INCREASING

With the more general recognition of this disease the number of reported cases is steadily increasing. The United States Public Health Service reports that thus far in 1929, 366 cases are on record. Cases of continued fever which are not obviously due to more common infections should be carefully studied so that this disease should not continue unrecognized.

TESTIMONIAL DINNER TO DR. LEARY

The Tufts College Medical School Alumni have completed preparations for a testimonial dinner to be tendered Dr. Timothy Leary at the Copley Plaza Hotel, Boston, on Wednesday, November 13, 1929, at 6:30 P. M. Dr. Leary recently resigned the Professorship of Pathology in the Medical School.

It is anticipated that this event will be the largest gathering of Tufts College Medical alumni in the history of the school.

Invited guests will include Governor Allen, Mayor Nichols, District Attorney Foley, President Cousens of Tufts College and Dr. A. Warren Stearns, Dean of the Medical School.

MASSACHUSETTS HOSPITALS APPROVED BY THE HOSPITAL STANDARDIZATION CONFERENCE OF THE AMERICAN COLLEGE OF SURGEONS

Ninety-three hospitals in Massachusetts have been given full approval by the Twelfth Annual Standardization Conference as follows:

Arlington, Symmes Arlington Hospital; Attleboro, Sturdy Memorial Hospital; Bedford, United States Veterans Hospital; Beverly, Beverly Hospital; Boston, Beth Israel Hospital, Boston City Hospital, Boston Dispensary Hospital for Children, Boston Lying-in Hospital, Carney Hospital, Children's and Infants' Hospital, Evangeline Booth Maternity Hospital and Home, Faulkner Hospital, Harley Private Hospital, House of the Good Samaritan, Long Island Hospital, Massachusetts Eye and Ear Infirmary, Massachusetts General Hospital, Massachusetts Homeopathic Hospital, New England Baptist Hospital, New England Hospital for Women and Children, Peter Bent Brigham Hospital, Robert Breck Brigham Hospital, St. Margaret's and St. Mary's Lying-in Hospitals, United States Marine Hospital; Brighton, St. Elizabeth's Hospital; Brockton, Brockton Hospital, Goddard Hospital; Brookline, Brooks Hospital, Free Hospital for Women; Cambridge, Cambridge City Hospital, Cambridge Hospital; Chelsea, Soldiers' Home in Massachusetts, United States Naval Hospital; Clinton, Clinton Hospital; Concord, Emerson Hospital in Concord; Fall River, Fall River General Hospital, St. Anne's Hospital, Truesdale Hospital, Union Hospital in Fall River; Fitchburg, Burbank Hospital; Framingham, Framingham-Union Hospital; Gardner, Henry Heywood Memorial Hospital; Greenfield, Franklin County Public Hospital; Haverhill, General Stephen Henry Gale Hospital; Holyoke, Holyoke Hospital, Providence Hospital; Lawrence, Lawrence General Hospital; Leominster, Leominster Hospital; Lowell, Lowell Corporation Hospital, Lowell General Hospital, St. John's Hospital; Lynn, Lynn Hospital; Malden, Malden Hospital; Medford, Lawrence Memorial Hospital; Melrose, Melrose Hospital, New England Sanitarium and Hospital; Milford, Milford Hospital; Montague City, Farren Memorial Hospital; Natick, Leonard Morse Hospital; New Bedford, St. Luke's Hospital; Newburyport, Anna Jaques Hospital; Newton Lower Falls, Newton Hospital; North Adams, North Adams Hospital; Northampton, Cooley Dickinson Hospital, United States Veterans Hospital; Norwood, Norwood Hospital; Peabody, Josiah B. Thomas Hospital; Pittsfield, House of Mercy Hospital, St. Luke's Hospital; Quincy, Quincy City Hospital; Roxbury, Hart Private Hospital, Roxbury Hospital; Rutland, United States Veterans Hospital; Salem, North Shore Babies' Hospital, Salem Hospital; Somerville, Somerville Hospital; Springfield, Mercy Hospital, Shriners' Hospital for Crippled Children, Springfield Hospital, Wesson Maternity Hospital, Wesson Memorial Hospital; Vineyard Haven, United States Marine Hospital; Waltham, Waltham Hospital; Westfield, Noble Hospital; Winchester, Winchester Hospital; Woburn, Charles Choate Memorial Hospital; Worcester, City Hospital, Fairlawn Hospital, Memorial Hospital, St. Vincent Hospital, Worcester Hahnemann Hospital.

COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT
WITH 1928 AND SEVEN YEAR AVERAGE MONTH
ENDING SEPTEMBER 28, 1929

DISEASES	1929					1928			
	Wk. ending Sept. 7	Wk. ending Sept. 14	Wk. ending Sept. 21	Wk. ending Sept. 28	Average cases reported for week corresponding to Sept. 28 for past seven years	Wk. ending Sept. 8	Wk. ending Sept. 15	Wk. ending Sept. 22	Wk. ending Sept. 29
Cerebrospinal Men.	-	-	-	-	-	-	1	1	-
Chickenpox	5	2	8	23	8	2	4	6	9
Diphtheria	4	19	18	10	28	16	10	26	17
Dysentery Bacillary	-	-	-	-	-	-	1	-	-
Encephalitis Epid.	-	-	-	-	-	-	-	1	-
German Measles	2	1	-	-	1	2	1	-	1
Influenza	1	1	1	1	3	2	3	8	2
Malaria	-	-	-	1	2	-	-	-	1
Measles	3	5	4	6	11	13	12	4	11
Mumps	3	6	4	5	7	5	10	4	15
Paratyphoid Fever	-	-	24	4	-	-	1	-	-
Pneumonia (Broncho)*	3	11	16	11	12	12	10	11	12
Pneumonia (Lobar)	6	9	6	7	14	7	13	13	11
Poliomyelitis	-	-	-	-	5	4	4	2	2
Scarlet Fever	5	7	9	23	27	3	6	15	14
Septic Sore Throat	-	1	1	-	-	-	1	-	1
Tetanus	1	-	-	-	-	-	-	-	-
Tuberculosis (Pul.)	14	26	36	34	30	42	29	28	16
Tuberculosis (O.F.)	1	3	1	-	2	4	1	-	-
Typhoid Fever	1	3	1	1	8	2	4	3	2
Undulant Fever**	-	2	-	1	-	-	1	-	-
Whooping Cough	18	17	31	15	33	43	37	27	20
Gonorrhoea	27	46	29	16	28	14	52	52	56
Syphilis	29	42	30	37	25	24	41	35	30

* Made reportable January 1, 1925.
** Made reportable July 1, 1928.

REMARKS: No cases of Asiatic cholera, Glanders, plague, rabies in humans and Yellow fever during the past seven years.

CORRESPONDENCE

A PLAN WHEREBY PATIENTS CAN BE SURE OF SERVICE

Mr. Editor—

William Bross Lloyd writes to you from 29 Newbury Street, complaining that in an emergency he telephoned to some twenty-five physicians before obtaining help, in many cases there being no answer from the doctor's office.

He thinks a little co-öperation among medical men would be a good thing.

If the physicians of Boston would do what has been done for years by those in this village, such troubles would be largely avoided.

A physicians' and surgeons' bureau is maintained here under the auspices of the Chamber of Commerce with twenty-four hour telephone service. A member who cannot for any reason have his telephone calls attended to reports to the bureau giving his temporary number. If he keeps his patients informed, he is therefore always in touch with them. Application to the Bureau direct also produces results for those who are unable to reach a selected physician, the attendant furnishing on request the names of those who can be reached at once.

Far be it for me to suggest that Boston might imitate Worcester yet there are a few good things even in this backward community.

Sincerely,

SAMUEL B. WOODWARD.

DR. CHEVALIER JACKSON OF PHILADELPHIA TO LECTURE AT BROCKTON

Editor, NEW ENGLAND JOURNAL OF MEDICINE:

On Tuesday, October 29th, at Massasoit Hall, 6 Main St., Brockton, at 8 P. M., Dr. Chevalier Jackson of Philadelphia, world renowned Bronchoscopist will lecture to the members of the Plymouth District.

The subject of his address will be: "Bronchoscopy as an Aid to the Internist and Surgeon in the Diagnosis and Treatment of Diseases of the Lung." Lantern slides and moving picture demonstrations.

We extend a cordial invitation to all physicians, members of the Massachusetts Medical Society, to be present as our guests.

Sincerely yours,
THOMAS H. MCCARTHY,
President, Plymouth District, Massachusetts Medical Society.

INFORMATION REGARDING OPPORTUNITIES FOR GENERAL PRACTICE

Tufts College Medical School
416 Huntington Avenue
Boston, Mass.

October 14, 1929.

Editor, NEW ENGLAND JOURNAL OF MEDICINE:

Information concerning attractive opportunities for general practice in New England is kept on file in the Dean's office at Tufts Medical School.

Yours very truly,
A. W. STEARNS, *Dean.*

PARKING PRIVILEGES IN NEW YORK

Editor, NEW ENGLAND JOURNAL OF MEDICINE:

October 21, 1929.

Dear Sir:

I am enclosing a clipping, copied below, from *The Diplomate* of September regarding the action taken in New York City concerning the parking of physicians' cars. I think it would be a good idea if something of this sort were taken up in Boston.

Yours very truly,
FRANK R. OBER.

PHYSICIANS ON DUTY MAY PARK IN NEW YORK CITY

Following conferences between a committee of the Medical Society of the County of New York and the Police Commissioner, it has been arranged that the police will recognize the automobile insignia issued by the American Medical Association, so that cars having this device will not be molested while the physician is making a professional call, even though his car is in a restricted area. The society is issuing cards to its members, so that the police may identify them should a question arise.

RECENT DEATHS

FITZGERALD—DR. JOHN JOSEPH FITZGERALD, a Fellow of the Massachusetts Medical Society since 1906, died at his home in Haverhill, October 19, 1929, aged 53.

He was a native of that city, was educated in the schools there, entered the drug business and then took the degree of M.D. from Baltimore Medical College in 1903. He had practiced the rest of his life in Haverhill and was school physician for six years. He was a member of the Knights of Columbus, the Haverhill Medical Club and Haverhill Country Club, and a Fellow of the American Medical Association. Dr. Fitzgerald is survived by his widow and two daughters.

COX—DR. SIMON FRANCIS COX, neuropsychiatrist of Lowell and Boston, died at the Ring Sanatorium, Arlington Heights, October 17, 1929, at the age of 55.

Dr. Cox was a graduate of Boston College in the class of 1896. His M.D. was from Harvard Medical School in 1900, subsequently serving as assistant superintendent at the Long Island Hospital, Boston Harbor.

Of late years Dr. Cox had been neuropsychiatrist to the Ring Sanatorium and superintendent of the Reconstruction Clinic, at 366 Commonwealth Avenue, Boston. He was a member of the New England Society of Neurology and Psychiatry and a Fellow of the Massachusetts Medical Society.

HARKINS—DR. JOHN FRANCIS HARKINS has died in Worcester, aged 54. He was born in Ireland January 8, 1875, and brought by his parents, the late William and Mary (Mulreany) Harkins, to Quincy, Massachusetts, at an early age. There he attended the schools with his brother, Dr. William J. Harkins of that city, a Fellow of the state medical society, and then went on to Holy Cross College, from which he received the degree of A.B. in 1896. Harvard University gave him an M.D. in 1900, and after study at the Johns Hopkins Medical School he settled in Worcester the following year and joined the Massachusetts Medical Society. Dr. Harkins held membership in: The Worcester Harvard Club, the Washington Club, Massachusetts Catholic Order of Foresters, Ancient Order of Hibernians and Knights of Columbus, being physician to the last named society for 25 years. He is survived by his widow, Cora Daniel Harkins.

SMITH—DR. PETER MATTHEW SMITH, a Fellow of the Massachusetts Medical Society, died at his home in Boston, October 13, 1929, at the age of 60.

He was a graduate of Georgetown University School of Medicine in 1894, settled in Boston and joined the State Medical Society three years later. At one time he was a Councilor from the Suffolk District.

Dr. Smith is survived by his widow, Hazel Moody Smith.

STEPHEN L. MALONEY

To those who are interested in public health of Boston the recent death of Stephen L. Maloney will be regarded as a great loss to the Boston Board of Health to which organization he gave loyal service as its Secretary. His influence was recognized in the affairs of the Massachusetts Association of Boards of Health. He was genial, energetic and a good public servant.

OBITUARIES

IN MEMORIAM

RANSOM HARVEY SARTWELL, M.D.

READ BEFORE THE MEMBERS OF THE NEW ENGLAND SOCIETY OF PSYCHIATRY AT THE MEETING HELD OCTOBER 8, 1929

Ransom Harvey Sartwell, M.D., was born the 30th day of December, 1887, in the town of Mooers, Clinton County, New York. He came of a sturdy stock. His father, Edwin Sartwell, and his mother, Olive Adsitt, were both natives of the town in which he was born. He was the youngest of a family of ten children. His preliminary education was received in the public schools of Mooers.

In 1911, at the age of 23, he was awarded the degree of Doctor of Medicine from the University of Vermont College of Medicine. Following his graduation he began the practice of his profession in Ellenburg, New York. After one year he removed to his native town, Mooers, where he practiced for two years. At the end of that period he accepted an appointment with an industrial concern located in Standish, New York.

Dr. Sartwell during his school work and following his graduation had gradually become interested in the field of psychiatry. He determined to enter this field, and with that idea in view, he applied for and received an appointment as Junior Assistant Physician at the State Hospital for Mental Diseases at Howard, Rhode Island. He entered upon his duties in August, 1915, where he remained until September, 1917. Later he transferred his activities from the State Hospital to the Penal and Reformatory Institutions of the State, he having been appointed as resident physician.

In March, 1918, he went to Foxboro, having accepted the position of Senior Assistant Physician at the Foxboro State Hospital. He remained there until September, 1923, when he was appointed Assistant Superintendent at the Worcester State Hospital. Dr. Sartwell remained at Worcester until August, 1924, when he was invited by the Director of State Institutions to return to Rhode Island, as Superintendent of the State Infirmary. Dr. Sartwell remained at the head of the Infirmary until his promotion to the Superintendency of the State Hospital for Mental Diseases at Howard, Rhode Island, October 1, 1926, which position he held at the time of his sudden death.

Dr. Sartwell was first married in 1920, and of this union there were two children, a son and daughter. His second marriage occurred in 1924.

Those of us who were associated closely with Dr. Sartwell can best realize what psychiatry and the community has lost in the death of this man. He was a faithful, conscientious physician, well grounded in medicine and had a lovable personality, a great consideration for the lives of others, and an intense loyalty to the organization he served. His vision was wide and he had great hopes of contributing to the advancement of psychiatry and indirectly as a result of this to the general happiness of human beings. By his death psychiatry has lost an able contributor, the medical profession an esteemed member and the community a good citizen.

THE BOSTON CITY HOSPITAL

In the death of Dr. Leon W. Jessaman we have lost both a fellow Alumnus and a faithful Staff Surgeon of the Boston City Hospital. To record formally such a loss as this in itself avails nothing, and yet, futile as it may seem, it helps us all, as we continue for a little while our earthly labors, to again appreciate his loyalty and devotion to our common endeavors. His example stimulates us to renewed efforts, for we know that he was one of those who "have left behind them that loving remembrance which is better than fame, and if their epitaphs are chiseled briefly in stone, they are written at full length on living tablets in a thousand homes to which they carried their ever welcome aid and sympathy".

October three
Nineteen hundred and twenty-nine

DR. JOSEPH W. COURTNEY

MEMORIAL RESOLUTION

READ BEFORE THE MEMBERS OF THE NEW ENGLAND SOCIETY OF PSYCHIATRY AT THE MEETING HELD OCTOBER 8, 1929

Whereas: In the death of Joseph W. Courtney, the New England Society of Psychiatry has lost one of its most notable and most active members. Having a brilliant, multi-facetted mind, he brought to the subject of psychiatry the neurological discipline of thought. Although thoroughly conversant with psychological literature, he was primarily a neurophysiologist in his approach to the theories of psychiatric thinking and research. A clear and logical thinker, a strong champion of his ideas, he was a much needed counterbalance to riotous theorizing in new fields of psychology. Dr. Courtney, who was an unusually cultured physician, fluently conversant with modern languages, a prolific reader of scientific and lay literature, an artist, a poet and a prose writer of ability, and charm, a delightful and forceful speaker with a vibrant, dynamic personality, added greatly to the prestige of our Society.

Therefore, Be It Resolved: That the New England Society of Psychiatry feels profoundly bereft in the loss of Joseph W. Courtney and deeply sympathizes with his family in their great loss.

NEWS ITEMS

GIFT TO NEWPORT, RHODE ISLAND, HOSPITAL—The Newport Rhode Island hospital has received two gifts of one hundred thousand dollars. The donors were Arthur Curtiss James and Lewis Cass Ledyard, both of New York.

These amounts are to be added to the permanent endowment fund which now becomes $315,000.

MADAME CURIE WILL AGAIN VISIT THE UNITED STATES—Madame Curie, referred to in the press as the world's most famous scientist, left Paris October 9 for a visit to this country.

American admirers of this celebrated woman will give her a second gram of radium.

Madame Curie will be a guest at the White House and will attend the dedication of Hepburn Hall of Chemistry, St. Lawrence University, Canton, New

York, and was present at the dinner celebrating the fiftieth anniversary of Edison's invention of the incandescent light. On the voyage the captain of the ship made preparations to avoid if possible any rough weather by changing the course of the vessel if warned by the wireless service. Fortunately the weather was propitious.

Governor Allen has extended an invitation to Mme. Curie to visit Massachusetts and observe the facilities for treating cancer in this Commonwealth.

NOTICES

REMOVAL

Dr. J. Harper Blaisdell announces the removal of his office to 5 Bay State Road, Boston.

Dr. Arthur T. Legg announces the removal of his office from 535 Beacon Street to the Longwood Medical Building, 319 Longwood Avenue, Boston.

REPORTS AND NOTICES OF MEETINGS

HARVARD MEDICAL SOCIETY

The first meeting of the Harvard Medical Society for the year 1929-30 was held on Tuesday evening, October 8, 1929 at 8:15 o'clock in the Peter Bent Brigham Hospital Amphitheatre. Following the customary presentation and discussion of cases, Dr. Harvey Cushing introduced Professor D. P. D. Wilkie of the University of Edinburgh who spoke on "The Etiology of Biliary Infections."

The first case was presented by Dr. Meagher. The patient was a 21 year old American-born typist whose family history and past history were essentially negative until two years ago when she had a left-tubo-ovarian abscess drained. Four months before admission she noted a painful, tender swelling in the left anterior triangle of the neck. An x-ray man advised her that she had bilateral cervical ribs and to think nothing of it. The tenderness continued and she entered the O. P. D. She gave a history of trouble in raising her arms. Examination showed the bilateral cervical ribs and what appeared to be a subclavian aneurysm. There were no neurological findings. In short, the patient had slight tenderness in the anterior triangles of the neck for four months, and for a year, periods of her left arms going to sleep. Examination shows bilateral cervical ribs and a probable subclavian aneurysm.

The second case was presented by Dr. Rennie of the medical service. The patient was a 39 year old Austrian-born insurance salesman who entered with the complaint of chills and fevers of five days' duration. His first entry in 1917 showed an enormous liver with tender nodules, a very large spleen, and a positive Wassermann. After antiluetic treatment he did not improve and was discharged with the diagnosis of biliary cirrhosis. He was tapped four times in 1917, each time removing 3-4 litres of fluid. In 1919 he had a Neisserian infection. In 1924 the question of splenectomy was raised, but his symptoms did not warrant the risk. For four years despite jaundice he remained well. In June 1929 he returned and salyrgen intravenously dissipated the fluid. Two days before the present admission he had a chill with a temperature of 103 degrees. Physical examination showed marked upward displacement of the thoracic organs with dulness at both bases. The abdomen was markedly distended and the spleen and liver were considerably enlarged. The fluid showed mainly red cells and had a specific gravity of 1.008. He had a mild anemia, a low white count, a slightly increased icteric index, and no bile in the urine. The diagnosis was Hanot's cirrhosis, associated, at present, with some local infection in the biliary tract.

In introducing his subject Professor Wilkie pointed out that what we know about biliary infections is mainly due to surgeons because the necropsy shows very little as the condition of the gall-bladder changes very quickly after death.

Concerning the anatomy of the gall-bladder, there are several important points. Considering its size the gall-bladder has a remarkably large blood supply and a large lymphatic drainage which terminates in the cystic gland at the gall-bladder neck. The nerve supply is through the vagus and the sympathetic; the sympathetic fibers extending through the hepatic to the coeliac plexus, and also to the phrenic plexus through fibers of the phrenic nerve. Finally, if the bile ducts are obstructed, the bile disappears and is replaced by the clear secretion of the mucosa.

There is still some question whether infection is always a precursor of gall-bladder disease. Because the cystic duct extends from the bottom of the gall-bladder, and a stone may lodge in the neck without entering the duct and a mucocele result. Then if secondary infection supervenes, a pus sac results. When this infection subsides and the gall-bladder relaxes, the stone may fall back, and repeat the story at a later date. The bile mixing with pus cells may cause the formation of gall-stones.

As to how this infection reaches the gall-bladder, four theories have been proposed: an ascending infection from the duodenum; infection from the liver through the bile; a spread to the gall-bladder through the lymphatics from a hepatitis; a blood-borne infection via the cystic artery. Experiments have been made to prove or disprove these theories. Of gall-bladders removed at operation, tissue cultures were made of the whole thickness of the wall, of the outer coat only, of the cystic gland, and of the bile. The bile never showed growth, the whole thickness only occasionally, and the other two almost always. It was very difficult to demonstrate the organisms even if the gall-bladder was obviously infected, yet sections of the wall showed the streptococci which previously had been obtained by culture. In 84% of cases an organism was found which while infecting the gall-bladder, paradoxically would not grow on bile. If these organisms were now injected into a healthy gall-bladder, a chronic cholecystitis resulted. To verify Rosenow's contention of an organism with an elective affinity for the gall-bladder, emulsions of cultures of these organisms were injected intravenously. In a few months the pathological picture of a chronic cholecystitis was obtained and, as in the other cases, with the formation of stones. The organisms were re-cultured from the gall-bladder walls. Furthermore, some animals were opened each week and the liver inspected. A hepatitis was found spreading from the gall-bladder. Finally, if the gall-bladder was separated from the liver by the omentum with no lymphatic connection, and the experiment

was done, cholecystitis and cholelithiasis, but no hepatitis resulted. The conclusions from these experiments were that gall-bladder disease is a streptococcus, blood-borne, intramural infection.

Professor Wilkie showed that the "strawberry" gall-bladder or cholesterosis is a direct antecedent of gall-stones. Lipoid polypi form on the lining mucosa, break off later on, and form pure cholesterin stones.

Professor Wilkie concluded that the gall-bladder may be a focus of body infection of great importance, and cited cases with remarkable results following cholecystectomy. His co-workers cultured streptococci from teeth abscesses and pyorrhea, and repeated their experiments. In only two cases did gall-bladder disease follow. Similar results were obtained with the tonsils. In spite of all these experiments, Professor Wilkie believes that the mouth is the primary source of infection for gall-bladder disease.

The treatment for gall-bladder trouble is obvious. If no natural resolution of the disease occurs, surgical removal of the gall-bladder should be done. The operation should not be limited to removal of the stone because the primary symptoms are from the gall-bladder and not from the stone.

The meeting was well attended and a more interesting paper has rarely been presented before the Society.

THE ANNUAL MEETING OF THE CAMBRIDGE TUBERCULOSIS ASSOCIATION

This meeting was held at the Phillips Brooks House October 17, 1929.

Reports of the year's work were submitted by Miss Florence C. Kelly, director of Sunshine Camp, and Mrs. Mabel Greeley Smith, executive secretary. Motion pictures of the camp were shown.

Dr. Cleaveland Floyd explained the application of business methods in fighting tuberculosis and other diseases and the relation of adequate income to health.

The officers for the ensuing year were elected as follows: Dr. Hilbert F. Day, who presided, was re-elected; Vice-presidents, Mrs. Rufus P. Williams and Dr. Felix McGirr; Secretary, Miss Louise W. Jackson; Treasurer, William D. Swan, Jr.

THE DUNHAM LECTURES

The Edward K. Dunham Lectures for the Promotion of the Medical Sciences for the year 1929-'30 were delivered at the Harvard Medical School amphitheatre, Building C. Joseph Barcroft, C.B.E., F.R.S., M.A., M.D. (hon.), Fellow of King's College, Cambridge, and Professor of Physiology, Cambridge University, spoke on the general subject of "Some Features in the Architecture of Function." Dr. W. B. Cannon introduced Professor Barcroft.

The title of the first lecture, given on Monday, October 7, was "Integrative Adaptation." Adaptation, said Professor Barcroft, is merely a process of integration, applied especially to body functions. He illustrated this by considering the changes in the blood during exercise. Four changes were dealt with: the oxygen content of the blood, the haemoglobin content, the rate of blood flow, and the so-called diffusion coefficient of the blood. These were discussed each in turn.

The second lecture on "The Constancy of the Internal Environment" was delivered on Wednesday, October 9. The thesis of this lecture was what man has gained by his constant internal environment over lower forms of life where such constancy is lacking. This was considered from several angles.

The temperature regulation of the heartbeat was investigated. This, unlike many other things, is not rigidly constant in man. In the frog it was found that the relation of the metabolism to the temperature is similar for the excised heart as for the whole frog. Thus, the velocity of these reactions and of all chemical reactions in the frog must be approximately the same to attain an equilibrium. Taking the equilibrium constant of haemoglobin as an example, that of man is five, while that of the frog is two. Therefore, it seems that an animal may assume an equilibrium constant compatible with life in its environment. If the temperature of a frog is increased, the pulse rate will also increase, but less and less for each increase of temperature. This same experiment was tried on a cat, both under light and deep anaesthesia. In a region of light anaesthesia the temperature coefficient seemed to be knocked out, so that as the temperature went down, the pulse went faster; at a still lower temperature it slowed down again. The experiment tried on man showed that as the external temperature decreased, the pulse went up. Then, at a point where shivering ceased and a pleasing sense of comfort pervaded the body of the subject, the pulse rate dropped to its initial slowness. The conclusions from this experiment were that during the "basking" period the normal temperature coefficient was operating, and that the fluctuation in the pulse rate was due to the shivering, acting through the sympathetic nervous system.

The H-ion concentration of the blood was also investigated as to its constancy. In the lower animals it is quite changeable. In the last analysis the regulation of the H-ion concentration depends on the respiration and through it on the respiratory centre. The respiratory centre seems to be diffuse. If the brain is sectioned beginning from the anterior end, nothing happens to the respiration until a section is made back of the corpora quadrigemina. Then, the normal or pneumotoxic breathing changes to apneustic breathing where large inspirations are divided by a few short ones. Then again, in the region of the 8th nerve the respirations become gasping in character and are unaffected by CO_2 changes in the blood, showing that buffering action no longer takes place. If the sectioning is continued, breathing ceases when the brain is cut at the calamus scriptorius.

What happens, then, queried Professor Barcroft, when constancy changes? If the temperature is low, inertia results; if high, delirium is seen. If the oxygen supply is deficient, unconsciousness results. A raised cH causes headache, if it is lowered, coma supervenes. A deficiency of sugar causes nervousness and hunger. An excess of water results in headache, nausea, dizziness, asthenia, inco-ördination. Too much salt gives reflex irritability, weakness and paresis, while a deficiency gives fever. If calcium is in excess, it causes apathy, drowsiness, coma, and

general atonia; if deficient, nervous twitchings and convulsions result.

In conclusion, Professor Barcroft speculated on why such relatively efficient organisms but with inconstant internal environments were not used as patterns for man. His conclusion was that organisms with inconstant body environments had sound bodies, but poorly developed minds, and that a constant internal environment is a necessity for a soundly functioning mentality of a higher type.

(To be continued)

THE NEW ENGLAND SOCIETY OF PSYCHIATRY

The Commonwealth of Massachusetts
Department of Mental Diseases
Grafton State Hospital
North Grafton, Mass.

October 18, 1929.

The semi-annual meeting of the New England Society of Psychiatry was held at the Danvers State Hospital on October 8. Dinner was served in the chapel, after which the members were welcomed by Dr. Clarence A. Bonner. In the absence of the President, Dr. John A. Houston, the Vice-President, Dr. Horace G. Ripley, superintendent of the Brattleboro Retreat, Vermont, presided. About 160 physicians interested in psychiatry attended.

Dr. Harry C. Solomon, of Boston, gave a "Moving Picture Demonstration of the Effect of the Inhalation of Carbon Dioxide in Certain Neuro-Psychiatric Conditions."

HARLAN L. PAINE, M.D., *Secretary-Treasurer.*

MASSACHUSETTS ASSOCIATION OF BOARDS OF HEALTH

More than one hundred and fifty health officers of eastern Massachusetts visited New Bedford on October 16 at the invitation of Mayor Ashley and the local board of health, the group being members of the Massachusetts Association of Boards of Health and the Associated Boards of Health of the Southeastern Health District. There were no business sessions, the purpose of the meeting being an inspection of the new "Superior" incinerator, "the only one of its kind in New England," and the screen house of the sewer system. This replaces the older system of outlets at different points along the wharves by an intercepting sewer, which discharges its contents half a mile down the bay. The city engineer explained the system to the visitors by means of charts and lantern views. At the clambake Mayor Ashley welcomed his guests and made a short reference to the necessity of spending public funds for health work. After dinner the company was entertained at the vast estate of Colonel E. H. R. Green in Dartmouth, where the "Mayflower," the baby "blimp," performed its evolutions and the old whaler, the Charles F. Morgan, was open for inspection.

MASSACHUSETTS DIETETIC ASSOCIATION

Women's Republican Club—46 Beacon Street, Boston

SCHEDULE OF MEETINGS

Tuesday, November 12, 8 P. M.
"Findings After Ten Years' Research in Poliomyelitis." Speaker, Harvard Infantile Paralysis Commission, Harvard University.
Business Meeting—Current Scientific Abstracts.

Tuesday, December 10, 8 P. M.
"The Growth of Nutrition in the Commercial World." Edith M. Barber, B.S., M.S., Writer and Consultant, Food and Nutrition, New York City.
Business Meeting.

Tuesday, January 14, 8 P. M.
"Metabolism of Inorganic Elements." Dr. L. T. Fairhall, Assistant Professor of Physiology, School of Public Health, Harvard University.
Business Meeting—Current Scientific Abstracts.

Tuesday, February 11, 6:30 P. M.
Dinner Meeting.
"The Most Recent Developments in Vitamin Investigation." Dr. Walter H. Eddy, Professor of Physiological Chemistry, Teachers' College, Columbia University, New York City.
Business Meeting.

Tuesday, March 11, 8 P. M.
"Nutrition from Public Health Standpoint." Dr. George Bigelow, Commissioner of Public Health, Commonwealth of Massachusetts.
Business Meeting.

Tuesday, March 25, 3 P. M. Field Trip.

Tuesday, April 8, 8 P. M.
"Influence of Canning Industry in the Changing Dietary." Dr. E. F. Kohman, Research Laboratories, National Canners' Association, Washington, D. C.
Business Meeting.

Tuesday, April 22, 3 P. M. Field Trip.

Tuesday, May 13, 8 P. M.
"Teas, Receptions and Dinners in Institutions." Alice Bradley, Principal, Fanny Farmer School of Cookery.
Business Meeting—Current Scientific Abstracts.

Tuesday, May 27, 8 P. M.
Annual Business Meeting—Election of Officers.

MASSACHUSETTS GENERAL HOSPITAL

The Suffolk District Medical Society will take over the Clinical Staff Meeting of the Massachusetts General Hospital, in the Moseley Memorial Building, on Wednesday, October 30, 1929, at 8:15 P. M.

PROGRAM

1. The Various Uses of Iodine in Goiter—Dr. J. H. Means.
2. An Unusual Genito-Urinary Problem—Dr. J. D. Barney.
3. Diaphragmatic Hernia in Secondary Anemia—Dr. A. V. Bock.
4. Interesting Gastro-Intestinal Cases—Dr. C. M. Jones.
5. Observations on a Group of Burn Cases—Dr. M. A. McIver.
6. Diagnostic Novocaine Block of Sympathetic Nerves—Dr. J. C. White.
7. Relief of Pain of Vascular Diseases of the Foot—Dr. R. Smithwick.

Doctors, medical students and nurses are cordially invited to attend.

COMMITTEE ON HOSPITAL MEETINGS.

THE INTERNATIONAL CONGRESS ON MENTAL HYGIENE—The first International Congress on Mental Hygiene which has been endorsed by a committee representing the mental hygiene movement throughout the world will be held in Washington, D. C., May 5-10, 1930.

President Hoover is Honorary President of the Congress and Dr. William A. White, Superintendent of St. Elizabeth's Hospital, Washington, is President. Clifford W. Beers is Secretary-General and Dr. Arthur H. Ruggles is Chairman of the Committee on Organization.

The American Psychiatric Association and the American Association for the Study of Feeble Mindedness will hold their annual meetings in association with the Congress.

National organizations interested in the various phases of mental hygiene will send representatives to Congress.

Administrative offices of the Congress are at 370 Seventh Avenue, New York City in charge of John R. Shillady, Administrative Secretary.

COMBINED MEETING OF THE PEDIATRIC SECTION OF THE NEW YORK ACADEMY OF MEDICINE, THE PHILADELPHIA PEDIATRIC SOCIETY, AND THE NEW ENGLAND PEDIATRIC SOCIETY

BOSTON, MASSACHUSETTS, OCTOBER 26, 1929

PROGRAM

9.30 A. M. to 12.30 P. M.

Children's Hospital Amphitheatre
300 Longwood Avenue, Boston

1. Impetigo Neonatorum: Its Prophylaxis in Maternity Hospitals, Orville R. Chadwell, M.D., Boston.
2. Acute Laryngitis with Obstruction, Edwin H. Place, M.D., Boston.
3. Intestinal Bacteria, Harold L. Higgins, M.D., Boston.
4. Tables of Average and Optimum Weight for Height, William R. P. Emerson, M.D., Boston.
5. Diagnosis and Prognosis, John Lovett Morse, M.D., Boston.
6. Some Deficiency Diseases with Especial Reference to Beriberi, Warren R. Sisson, M.D., Boston.
7. A Milk-free Food for Infants with Milk Idiosyncrasy, Lewis Webb Hill, M.D., Boston.
8. Demonstration of Artificial Respiration Apparatus, James Wilson, M.D., Boston.
9. Hyperpnea, James L. Gamble, M.D., Boston.
10. Some Observations on the Treatment of Hemophilia, Stewart Clifford, M.D., Boston, Cannon Ely, M.D., Boston.
11. Demonstration of Cases, Bronson Crothers, M.D., Boston.

1.00 to 2.00 P. M.

Buffet luncheon in Vanderbilt Hall.

2.00 to 3.00 P. M.

Children's Hospital Amphitheatre

1. Weight, Growth and Nutrition, Raymond Franzen, Ph.D., New York City.

After the meeting Dr. Ober will be glad to meet anyone who wishes to visit the Peabody Home for Crippled Children or

Dr. Talbot's and Dr. Higgins' laboratory at the Massachusetts General Hospital will be open to anyone who wishes to see the demonstration of the Metabolism Apparatus.

7.15 P. M.

Dinner at the Kenmore, 496 Commonwealth Avenue, Boston.

Commander Donald MacMillan of Wiscasset, Maine, will give an informal talk on "Work in the Far North."

GENERAL INFORMATION

Breakfast will be served at 8.30 A. M. to the visiting guests of the Society at the University Club, 40 Trinity Place, Boston, Massachusetts. The University Club is one block north from the Back Bay Station.

For Children's Hospital take any outward bound Huntington Avenue car and get off at Longwood Avenue and walk two blocks west.

Dinner tickets may be obtained from the Secretary of the New England Pediatric Society by mail or at the time of the dinner. It is important that we know as early as possible how many will attend the luncheon and the dinner. Price $4.00. Dress will be informal.

SUFFOLK DISTRICT MEDICAL SOCIETY

CENSORS' MEETING

The Censors of the Suffolk District Medical Society will meet for the examination of candidates at the Medical Library, No. 8 The Fenway, Thursday, November 7, 1929, at 4:00 o'clock.

Candidates should make personal application to the Secretary and present their medical diploma at least one week before the examination.

LELAND S. MCKITTRICK, *Secretary.*

205 Beacon Street, Boston, Mass.

AMERICAN COLLEGE OF PHYSICIANS

The Fourteenth Annual Clinical Session of the American College of Physicians will be held at Minneapolis, Minn., February 10-14, 1930.

Headquarters—Minneapolis Municipal Auditorium.

Hotels—Curtis, Leamington, Radisson, Sheridan and others.

Watch monthly announcements in *Annals of Internal Medicine.*

John H. Musser, M.D., President, New Orleans, La.

S. Marx White, M.D., General Chairman, Minneapolis, Minn.

E. R. LOVELAND, *Executive Secretary,*
133-135 S. 36th St., Philadelphia, Pa.

INTERNATIONAL MEDICAL POSTGRADUATE COURSES IN BERLIN

are arranged with the help of the medical faculty of the University by the Lecturers' Association for medical continuation courses and the Kaiserin Friedrich-Haus. Part of the courses take place permanently, part only in March and October, 1930.

I. Permanent Courses

a) of monthly and semi-monthly duration, dealing with all kinds of branches of medicine.

b) Courses as guest-assistants in clinics, hospitals and laboratories during two to three months and longer for gentlemen desiring to do practical work under systematic supervision.

II. Courses in March, 1930

1. Group course: Survey of modern medical science (actual questions relating to internal diagnostics and therapeutics), (from March 3-15), fee RM 75.

2. Postgraduate course covering the domain of skin and venereal diseases (from March 17-29), fee RM 100.

3. Obstetrical-gynaecological postgraduate week (from March 24-29), fee RM 50.

4. Fifth postgraduate course on practical improvements in ray-therapeutics (from March 30-April 7), fee RM 100.

5. Special course for surgery (from April 28-May 3), fee RM 75.

6. Single courses on all special fields of medical science including practical work.

III. Courses in October, 1930

For October, 1930, the following courses are planned:

A course "practical laboratory".

A children's course.

A course on internal medicine.

An oculist course.

A course on trade medical science, insurance medical science and experts' profession.

The courses are held in German, but numerous professors are able to lecture in the English, French and Spanish languages.

The information bureau of the Kaiserin Friedrich-Haus für das ärztliche Fortbildungswesen, Berlin NW 6, Luisenplatz 2-4, instrumental in procuring suitable lodgings, gives information as to cost of stay, arranges the attendance in clinics at operations, etc., and, upon desire, sends detailed syllabuses.

BRISTOL COUNTY ORTHOPAEDIC CLUB

The Clinical Meeting of the Bristol County Orthopaedic Club will be held at St. Luke's Hospital, Allen Street, New Bedford, Mass., October 29, 1929 at 8:15 P. M.

Earle E. Hussey, M.D., *Secretary.*

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held at 8.15 on the evening of Tuesday, October 29, in the amphitheatre of the Peter Bent Brigham Hospital. Program:

1. Presentation of Cases, one of which will be a demonstration of prolonged artificial respiration in a case of Poliomyelitis.

2. Recent Studies in Epilepsy. Dr. William G. Lennox.

Dr. William P. Murphy, *Secretary.*

THE ANNUAL MEETING OF THE MASSACHUSETTS PSYCHIATRIC SOCIETY

The annual meeting of the Massachusetts Psychiatric Society will be held at the Hotel Somerset on Wednesday evening, October 30, at 6.30 P. M.

The speaker of the evening will be Dean Milton C. Winternitz of the Yale Medical School, who will take as his topic "The Institute of Human Relations".

Winfred Overholser, *Secretary.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

October 25—Massachusetts Homoeopathic Hospital. Detailed notice appears on page 796, issue of October 17.

October 26—Combined meeting of the Pediatric Section of the New York Academy of Medicine, the Philadelphia Pediatric Society, and the New England Pediatric Society. Detailed notice appears on page 853.

October 26—The Section of Pediatrics of the New York Academy of Medicine. Complete notice appears on page 796, issue of October 17.

October 29—Bristol County Orthopaedic Club. Detailed notice appears elsewhere on this page.

October 29—Harvard Medical Society. Detailed notice appears elsewhere on this page.

October 30—Massachusetts General Hospital. Detailed notice appears on page 852.

October 30—The Annual Meeting of the Massachusetts Psychiatric Society. Detailed notice appears above.

1929-1930—Massachusetts Dietetic Association. Complete schedule appears on page 852.

November 4, 5, 6 and 7, 1929—Clinical Congress and Eighth Annual Meeting of the American College of Physical Therapy. See page 149, issue of July 18, for details.

December 2-6—The Radiological Society of North America. Complete notice appears on page 149, issue of July 18.

February 10-14, 1930—American College of Physicians. Complete notice appears on page 853.

May 5-10, 1930—The International Congress on Mental Hygiene. Detailed notice appears on page 853.

DISTRICT MEDICAL SOCIETIES

Berkshire County Medical Society

October 30—The next meeting of this Society will be held at the Park Club, Pittsfield, at two o'clock.

The speaker will be Dr. Chevalier Jackson of Philadelphia.

The subject will be: "Prevention of a Certain Class of Accidents to Children." This meeting will be for semi-professional workers, child-welfare organizations, as well as hospital authorities and student nurses. An invitation is also extended to the laity who might be interested.

HUGH DOWNEY, Secretary.

Essex North District Medical Society

November 7 — Thursday — Censors' meeting at Hotel Bartlett, 95 Main Street, Haverhill, (Telephone 3430) at 2 P. M. sharp. Candidates should present their diplomas to the Secretary one week in advance.

January 8, 1930—Wednesday—Semi-annual meeting at Centre Church Vestries, Main Street, corner of Vestry Street, opposite City Hall, Haverhill, (Telephone 548) at 12:30 sharp.

May 1—Thursday—Censors' meeting at Hotel Bartlett, 95 Main Street, Haverhill, (Telephone 3430) at 2 P. M. sharp. Candidates should present their diplomas to the Secretary one week in advance.

May 7—Wednesday—Annual Meeting.

J. FORREST BURNHAM, Secretary.

Franklin District Medical Society

The meetings of the Franklin District Medical Society will be held at the Weldon Hotel, Greenfield, Mass., on the second Tuesdays of November, January, March and May, at 11 A. M.

CHARLES MOLINE, Secretary.

Hampshire District Medical Society

The meetings of Hampshire District will occur at 11 A. M. the second Wednesdays of December, February, and April and will be held at the Dickinson Hospital, Northampton. The annual meeting will be held in May.

LUTHER O. WHITMAN, M.D., Secretary.

Middlesex East District Medical Society

November 13—At Reading.

January 15—At Boston, Harvard Club.

March 12—At Melrose.

May 13—At Unicorn Country Club.

ALLAN R. CUNNINGHAM, Secretary.

Middlesex South District Medical Society

October 29—This meeting comes at the invitation of Plymouth District Medical Society, to be held at Massasoit Hall, 6 Main Street, Brockton, at 8 P. M. Dr. Chevalier Jackson of Philadelphia will give an address, "Bronchoscopy as an Aid to the Internist and Surgeon in the Diagnosis and Treatment of Diseases of the Lung." Lantern slides and moving picture demonstrations.

November 6—Censors' meeting.

April 16, 1930—Annual meeting. Commander Hotel, Cambridge. Program to be announced.

Winter meetings—jointly with Suffolk District.

May, 1930—Censors' meeting.

ALEXANDER A. LEVI, Secretary.

Norfolk District Medical Society

A regular meeting of the Society will be held in the Medfield State Hospital, Harding, Mass., October 29, 1929.

Program:

The afternoon will be devoted to inspection of the hospital. Members will be conducted about in groups. Dr. E. H. Cohoon, Superintendent of the hospital, requests that members plan to arrive as early as is possible, for the hospital is large and considerable time will be required to see even a small part of it.

5:30 P. M. Business Meeting.

6:00 P. M. Luncheon.

Communications: Immediately following the luncheon.

Elisha H. Cohoon, M.D.
"The State Mental Hospital and Its Associated Activities."

Samuel Smith Cottrell, M.D.
"Postural Sequelae of Encephalitis Lethargica."

Your executive committee urgently requests that all possible attend this meeting. Out-of-town meetings have in the past proven popular and profitable. Shall we continue them?

Medfield State Hospital may be reached from Boston by several routes: either through Wellesley, turning off at South Natick, through Dover to hospital; or through West Roxbury, Dedham, Westwood to Medfield. All distances are about the same. The distance from the hospital to the State House is approximately 21½ miles. All roads are good.

FRANK S. CRUICKSHANK, M.D., Secretary,
47 Bay State Road, Boston.

November 25—Roxbury Masonic Temple. Dr. Roger Graves, "Urology from the Standpoint of the General Practitioner." Surgical discussion. Medical discussion.

January 27, 1930—Roxbury Masonic Temple. "Recent Advances in Diagnosis and Treatment of Syphilis." The speakers will be Drs. C. Morton Smith and Austin W. Cheever.

February 24, 1930—Roxbury Masonic Temple, 8:00. Dr. Edwin H. Place has been invited to read a paper and has been given carte blanche in the selection of his subject.

March 24, 1930—Roxbury Masonic Temple. Dr. Burton E. Hamilton. Subject to be announced.

May 6, 1930—Annual Meeting. Program to be announced.

The Censors meet for the examination of candidates, November 7, 1929, and May 1, 1930, in the Roxbury Masonic Temple, 171 Warren Street, Roxbury, at 4:00 P. M.

Applications must be in the hands of the Secretary at least one week previous to date of examination.

Owing to inability to engage the Roxbury Masonic Apartments on the last Tuesday of the month, such meetings as will be held in the Roxbury Temple will be held Monday evenings as per the dates above.

FRANK S. CRUICKSHANK,
Secretary, Norfolk District Medical Society.

Plymouth District Medical Society

October 29—Massasoit Hall, 6 Main Street, Brockton, at 8 P. M. Speaker, Dr. Chevalier Jackson of Philadelphia. Subject: "Bronchoscopy as an Aid to the Internist and Surgeon in the Diagnosis and Treatment of Diseases of the Lung." Lantern slides and moving picture demonstration.

All physicians, members of the Massachusetts Medical Society are cordially invited to be present. **This is an important meeting.**

THOMAS H. McCARTHY, President.

Suffolk District Medical Society

October 30—Massachusetts General Hospital. Detailed notice appears on page 852.

November 7—Censors' meeting. Detailed notice appears on page 853.

BOOK REVIEWS

Minor Surgery. By FREDERICK CHRISTOPHER. W. B. Saunders Co., Philadelphia. 1929. 694 pages. Price $8.00.

This volume covers the field of minor surgery in a lucid comprehensive manner, carefully avoiding encroachment on major work and undue detail in the very simple phases. As brought out by Allen B. Kanavel in the foreword, this book fills an important need in the department of surgery comprising the greatest number of cases yet usually receiving but slight consideration in the medical curriculum. Most of the material is taken from Christopher's own wide experience, but very generous use is made of the work of others, nearly every page containing a reference to some recent original article where the reader may secure details of the topic under discussion. The large number of illustrations add greatly to the value of the text.

After a general consideration of wounds, foreign bodies, infections, burns, electrical injuries, and circulatory disturbances, the subject is covered on an anatomical basis. With such a well balanced work it is difficult to select any portion for special comment, yet the discussion of carbuncles, of the injection treatment of varicose veins, and of back injuries is so complete and still concise, that it should prove of especial value. The ability of Christopher to cover all the important phases of a subject in a practical manner with relatively few words is illustrated by the sections on fractures and on infections. The volume closes with a section addressed to the surgical internes and replete with valuable suggestions on the many details of the interne's duties, including post-operative care.

As a whole this book impresses the reviewer as the most valuable aid in the treatment of minor surgical conditions that he has yet seen.

Experiments and Observations on the Gastric Juice and the Physiology of Digestion. By WILLIAM BEAUMONT, M.D., facsimile of the original edition of 1833, together with a biographical essay, *A Pioneer American Physiologist*, by SIR WILLIAM OSLER. Cambridge: Harvard University Press, 1929. xl + 280 pages. Price, $3.00.

In connection with the meeting of the 13th International Physiological Congress held in Boston in August of this year, this volume has been issued. It is a careful facsimile reprint of Beaumont's original work, first published in 1833. In addition there is reprinted also the familiar biographical essay on Beaumont, "A Pioneer American Physiologist" by Sir William Osler, first read before the St. Louis Medical Society, October 4, 1902. Copies of the

first edition of Beaumont's work are exceedingly scarce and a careful reprint is welcome. It is one of the outstanding contributions to medicine made by this country and some would argue with good reason that it is a far more important contribution than any other that has been made. The book is beautifully printed and a great credit to the publisher.

The Principles of Clinical Pathology in Practice. By GEOFFREY BOURNE, M.D., and KENNETH STONE, M.D. Oxford University Press. London. 1929. Pp. 392. Price $4.75.

This is a useful summary of the various points in clinical pathology that aid in diagnosis. It is not a technical volume in the sense of giving methods. It emphasizes more the standpoint of the diagnostic value of the various information gleaned by laboratory tests. It serves more as a guide for the practitioner of medicine in his efforts to make diagnosis than it does as a guide for the laboratory worker. The chief effort is to enumerate under the various headings the types of tests which are of value for diagnosis.

The Diagnosis of Pancreatic Disease, by ROBERT COOPE, M.D., B.Sc., M.R.C.P., Senior Assistant Physician, Royal Southern Hospital, Liverpool. Oxford University Press. 112 pages. Price $1.50.

To one interested in a clear, but rather academic discussion of one of the knotty diagnostic problems of internal medicine this little monograph is worthy of perusal. It takes up in sufficient detail the anatomy, physiology, and pathology of pancreatic disease. The symptomatology of acute disease of the organ is fully and accurately described. The chronic involvement of the gland is of necessity described in a very general manner. Turning from clinical considerations to laboratory tests of pancreatic function, the author displays an attitude of refreshing and healthy scepticism of practically all qualitative or quantitative tests and presents the appropriate evidence for his disbeliefs. In general he attempts to simplify existing tests and to discard all but those that have practical significance. In so doing he clears the air of many pseudoscientific misconceptions and emphasizes impressively the many difficulties that are involved in consideration of disease of the pancreas. One pertinent fact he stresses, namely, "diagnosis of pancreatic lesions would be easier if the doctor remembered that his patient had a pancreas". The writer is to be commended for his brevity and his intelligent presentation of a difficult subject. There is a very excellent bibliography.

The History of Nursing. By JAMES J. WALSH, M.D., Ph.D. P. J. Kenedy and Sons. New York. 1929. Pp. 293. Price $2.15 post paid.

To those whose history of nursing begins with Florence Nightingale and the Crimean War this volume will be of much interest in that it traces the development of the nursing sisterhood from the early days to the present. There is a chapter on nursing in the days of primitive Christianity and then the development of hospitals and the system of caring for the patients in the days of the Roman Empire.

Still another interesting section is on the leper hospitals and the rôle which they played in the control of leprosy in Europe.

A considerable portion of the book is naturally devoted to nursing in America, divided into the Spanish phase, the French phase, the period of hospital decadence, and then the development of the Sisters of Charity and thereafter the development of the modern trained nurse.

An Introduction to Pharmacology and Therapeutics. By J. A. GUNN, M.D., D.Sc. (Edin.); Professor of Pharmacology in the University of Oxford and Fellow of Balliol College; sometime Examiner in the Universities of Belfast, Bristol, Cambridge, Cardiff, Edinburgh, Leeds, Liverpool, London, Oxford, Sheffield, and to **the Royal College of** Physicians. Humphrey Milford. Oxford University Press. London, New York, etc. 1929. Price $1.50.

This small volume (12 mo. 220 pp.) is an excellent exposition of its subject, based upon its author's lectures to medical students. It can be recommended highly as a general survey of pharmacology and pharmacotherapeutics for use in medical courses where the actual treatment of patients and their diseases is taught clinically after such an introduction. It includes brief but adequate descriptions of all of the drugs in common use. The principal official preparations and their dosage are given both according to British and to the United States Pharmacopeiae.

Pathogenic Micro-organism. By WILLIAM H. PARK, M.D., A. W. WILLIAMS, M.D., and CHARLES KRUMWIEDE, M.D. 9th Edition. Lea & Febiger. Philadelphia. 1929. Pp. 819. Price $6.50.

A new edition of Dr. Park's book needs no introduction. The pioneer work which he and his coworkers have done in advancing our knowledge and our means of control of the bacteria have been incorporated in this book which started as a manual, primarily for the use of the practitioners of medicine, based on the procedures in use in the laboratory of the City of New York. In this edition the sections on scarlet fever, tularemia and undulant fever are of special interest. The section on pneumococcus typing has been revised and there is brief mention of the B. C. G. method of vaccination against tuberculosis.

There is no other volume of this type that includes as much technical, practical and theoretical information in so concise a form.